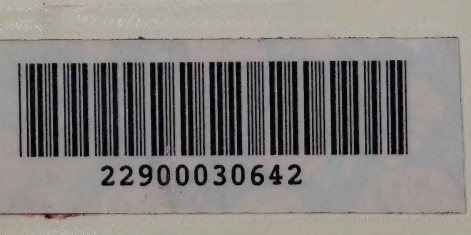
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TWENTIETH CENTURY PRACTICE

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IN TWENTY VOLUMES

VOLUME IV.

DISEASES OF THE VASCULAR SYSTEM AND THYROID GLAND.

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DISEASES

OF THE

HEART AND PERICARDIUM.

BY

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DISEASES OF THE HEART AND PERICARDIUM.

HISTORICAL NOTE.

THERE was no knowledge of disease of the heart in antiquity. Hippocrates did not believe that the heart could be diseased, and this view prevailed as late as the time of Celsus. The heart was the centre of life, and disease of the heart was incompatible with life itself. The symptoms of heart disease which are now considered as characteristic, to wit, palpitation, dyspnœa, cyanosis, were interpreted as evidence of disease of the lungs, of the nerves, etc.

The first intimations of heart disease date from the day of Galen, 131–201 A.D., whose ideas were based wholly upon the dissection of animals. Galen located the heart in the middle of the chest. He was quite well acquainted with its structure and described the oblique course of its fibres. He was familiar with pericarditis, with effusions, exudations, and so-called polypi, and maintained that these conditions might occur in man. But these views, because undemonstrable, passed unregarded. Galen first showed that the arteries contain blood, though he still admitted a mixture of air with the blood. The useful part of the blood was believed to traverse the ventricular septum through orifices to the left heart, while the useless part was conveyed by the pulmonary artery to the lungs whence it was exhaled. The pulmonary veins carried to the left heart the air (pneuma) which was mixed with the blood.

Galen maintained that the heart contains within itself the source of its own motion, as he saw it continue to beat after separation from all its vessels. Unfortunately, Galen believed in a peculiar principle of life, a belief of which many physiologists of the present day are not yet entirely disembarrassed. Galen located the seat of this principle in the heart, and was thus robbed of all the profits of his anatomical studies. These views prevailed undisturbed for over a thousand years, until the great anatomist of the Sixteenth Century, Vesalius (1543), with true descriptions of the construction of the heart of man, cleared them away as fallacies.

Benivieni (1506) had already found in the condition of the heart the cause of certain cases of sudden death, but there could be no real knowledge of the nature of heart disease until at least the course of the blood through its various chambers had been established. But, strange to say, this unapproachable discovery by Harvey (1628) produced no immediate effect upon practice, which remained based almost wholly upon symptomatology and empiricism, and it was only after the lapse of another century that clinicians were enabled to interpret heart disease through the knowledge of the circulation of the blood.

Thereupon Vieussens (1685) described a case of sclerosis with insufficiency of the mitral valve connected with hypertrophy. Lancisi (1707) observed dilatation of the right side of the heart, and called attention to the undulations of the cervical veins. Albertini emphasized the fact that while the left ventricle more frequently undergoes hypertrophy, the right sooner suffers dilatation. These affections are revealed in life by palpitation and angina, and are frequent causes of sudden death. More distinctly and fully Morgagni (1761) ("De Sedibus et Causis Morborum") described the mechanism of valve lesions and derived cyanosis from defective aëration of the blood. Morgagni also saw brain disease supervene as a consequence of disease of the heart; described pericardial effusions; noticed the adhesion of the two layers with obliteration of the sac, and mentioned in detail the changes which take place in dilatation of the aorta, aneurysm. Sénac (1749) busied himself with the clinical aspects of heart disease, recognizing prominence of the anterior chest-wall as an evidence of enlargement of the heart. Sénac was aware also of the connection of heart and brain disease, and knew something of the changes of age.

The means of examination were as yet too few for further diagnosis. About this time Auenbrugger (1761) published his small book on percussion, worked out, he declares, during the course of seven years, *inter tœdia et labores*, which was to subsequently illuminate a wide field of heart disease. Auenbrugger called attention to the increased area of dulness in cases of pericardial effusion and enlargement of the heart, but as has so often been remarked in the history of discovery, this new invention, which was the most remarkable contribution of the age, attracted no attention whatever. The author was a simple practitioner without position or name, and his discovery, which was finally to work a revolution in the study of heart disease, was mentioned by the few who noticed it at all with derision and contempt. Corvisart exhumed the book, translated it into French (1811), and made it public property. Corvisart declared that, by the use of percussion, he could estimate the diameters and circumferences

of the heart, whereby he was able to recognize pericarditis and to differentiate hypertrophies from dilatations. Corvisart thus clearly established the relation between valve lesions and enlargements. Now, although the full value of percussion remained to be appreciated only by Piorri (1840), the method of study was at once adopted throughout France and England. Laennec declared that Corvisart was the first to feel the frémissement cataire in valve disease.

Corvisart described vegetations upon the valves, which he denominated verrucose, because they were like the venereal excrescences or warts which develop upon the glans, vulva, and nymphæ. Corvisart considered them of venereal origin, a view which was rejected by his pupil, the great Laennec, for the naïve reason that the venereal affections are too frequent and vegetations too few. The vegetations are encountered also, he declares, in subjects free from any venereal disease.

Laennec (1819) is the undisputed author of auscultation, which as by an electric light illuminated the darkest recesses of heart disease. Palpation had been used by Hippocrates, and it was his custom, as it was that of his followers, to place the hand on the heart in appreciation of its tumultuous beatings, muffling, or location. Some intimation of percussion can be found in the works of Hippocrates and subsequent writers, though it had no real value until published by Auenbrugger and no recognized value until proclaimed in the translation of his work by Corvisart. But there are no previous intimations regarding auscultation and, with the exception of a single statement by Horne Tooke, since exhumed, which likened the sound of internal organs to the whirr of machinery or the sounds of a clock, and which this author timidly ventured to hope might prove of value in the determination of diseases of the chest, there is no mention made of auscultation until the time of Laennec. Laennec invented the stethoscope, with which he distinguished forms of valve disease in life, and concentrated the attention of subsequent observers upon the study of diseases of the heart.

Since the disclosure of the anatomy of the heart by Vesalius, and the revelation of the circulation of the blood by Harvey, no subsequent discovery in the field of heart disease could ever in any way be compared with the contributions of Laennec. These are the three great lights in anatomy, physiology, and pathology, which have rendered it possible for subsequent observers to see.

Next, Richard Bright (1827) discovered the enlargement of the heart which occurs in kidney disease, and this subject was especially elaborated later by Traube (1856). Pericarditis was known to Galen. It was reserved for Kreysig and Bouillaud (1830) to make the first

recognition of endocarditis. Virchow (1856) developed the doctrine of thrombus and embolus and thus cleared up certain hitherto inexplicable complications of heart disease on the part of the brain, lungs, and kidneys.

The subsequent great epoch in the history of heart disease lies largely in the field of etiology, in the discovery of the relation of the infections and the rôle of micro-organisms in the production of lesions. These discoveries belong to the present time and are described in the study of the etiology and pathology of the various lesions.

DISEASES OF THE PERICARDIUM.

Absence or Defect.

Congenital anomalies of the pericardium are more rare than those of the heart itself. Concretion of the two layers has been observed in the fœtus and at birth. This subject receives special consideration. Still more infrequent are defects in the pericardium. But sometimes the pericardium is entirely absent, or it may be reduced to a few fibres at the base of the heart. Defects when they occur are more commonly partial. Sometimes the membrane shows slits, through which the heart may protrude into the left pleural cavity. Sometimes the heart lies out in the mediastinum; sometimes it lies with the lung in the common sac formed by the pleura while the visceral layer of the pericardium remains a direct envelope of the heart. Hüter reported a case of adhesion of the pericardium with the heart during uterine life. Curling and Pisek described cases of congenital absence of the pericardium. Baly reported a case of absence of the pericardial sac, in which the heart was found lying in the cavity of the left pleura, and Chiari a case of almost perfect defect of the parietal pericardium. Carter described a case of hernia into the pericardium, and Powell a case of pneumothorax with congenital opening in the pericardium. Weisbach saw a congenital defect of the pericardium, and Turner showed a case in which the pericardium was unattached to the diaphragm.

Defects of the pericardium may not be recognized in life, though Faber thought that the condition might be surmised on account of the stronger mobility of the heart and the delay in the apex stroke.

Diverticula are sometimes reported: thus Bristowe and Cuffer reported cases of diverticula of the pericardium. Hart described a case in which a diverticulum contained three to four ounces of fluid. Baudy, of St. Louis, reported a case of cystoid diverticulum of the pericardium, and Speir showed to the Medical Society of the County of Kings, New York, a specimen of such diverticulum.

Diverticula are usually developed under pressure of fluid from within. The fibrous layer yields, becomes thinned or splits under the pressure to allow the serous layer to protrude through it like a pocket. Cruveilhier reported a case in which a diverticulum was caused by the pressure of blood which had issued from a rupture of the left ventricle.

It may be readily understood that a diverticulum might form an embarrassing element in the diagnosis of pericardial effusion.

Tumors.

Ecchymoses of the pericardium are sometimes seen in diseases attended by disorganization of the blood, as in scurvy, leucæmia, anæmia, purpura, etc., as also occasionally in phosphorus-poisoning. More extensive extravasations have been observed under the extreme venous stasis of heart disease, in death by suffocation, etc. The opacities which are observed disseminated over the pericardium, especially on the visceral layer constituting the so-called milk spots, are studied in connection with pericarditis.

Neoplasms are exceedingly rare, but occasional cases can be found scattered in medical literature. Thus Bouchard reported a case of polypi of the pericardium in an infant aged four; Hohnbaum a similar case in more advanced life. Free bodies, which are supposed to have originated from polypi, have sometimes been found in the pericardium. They vary in consistence, being sometimes soft and smooth like epithelial tissue, sometimes hard like fibroid tissue, sometimes stratified, occasionally calcified to constitute cardiac calculi (cardioliths). Bouchard found synovial fringes, sometimes pedunculated, sometimes detached so as to constitute foreign bodies.

Hydatids of the pericardium are rare, but one case of this kind was reported by Barlow. Enos and Rapp described cases of cyst of the pericardium, and Chadzynski reported a case of enormous hydatid in the interior of the pericardium which caused sudden death, but showed during the whole period of its development no clinical evidence whatever. Bernheim cites five cases of hydatids of the pericardium usually coexisting with hydatids of other organs. A case of hydatid of the pericardium, lungs, and liver, requiring two evacuations by the use of drainage-tubes, and finally aspiration, was reported by MacDonald. The case had been diagnosticated as tuberculosis with a fatal prognosis. A fetid cyst communicated with a bronchial tube in the infra-axillary region, and another was drained in the infra-clavicular region of the left lung. Aspiration discharged another cyst over the apex of the heart (Gaston). Hydatids might be suspected when the symptoms of heart disease ensue in the course

of echinococcus of the liver and other organs. The condition is so rare as to constitute what Petit calls "a surprise at the autopsy." Reports of the finding of cysticercus and trichina may be found among the curiosities in the literature of the pericardium.

The finding of fibroid tumors and lymphomata was reported by Chambers and Bernheim. Ullé once encountered a cystic enchondroma in the pericardium.

Cancer.

The pericardium is an infrequent site of malignant disease. Such deposits are usually metastatic. Primary deposits imply affection of the lymph glands of the pericardium. Guarnieri reported a case of endothelial cancer primary in the pericardium. Marchiafava described a case of endothelioma primary in the lymphatics of the pericardium, and Liborius a case of sarcoma of the pericardium and bronchial glands. Bernheim cites two cases of primary cancer of the pericardium reported by Foerster and Le Beef. Secondary cancer is but little more frequent. Willigk found the pericardium the seat of cancer seven times in 477 cases of various kinds of cancer. The cancer extends in these cases from some contiguous viscus, the mediastinum, bronchial glands, pleura, lungs, œsophagus, etc. (Doléris, Barth, Liborius), sometimes from more remote origin (Cruveilhier, Viguier, Clay). The condition is surmised by the presence of cancer elsewhere, by the occurrence of enlarged glands in the neighborhood, as in the subclavicular region. The fluid discharged by aspiration is usually bloody and is sometimes ichorous. Nicholls saw a case of malignant disease of the pericardium which simulated aneurysm of the aorta.

Balzar reported a case of miliary aneurysm of the pericardium in a syphilitic subject.

Syphilis.

Syphilis usually spares the heart, or if it attack the heart spares the pericardium. Of 110 cases collected from the literature by Mracek, there was but one case of gummatous pericarditis, one of associate peri- and endocarditis and six of all organic parts of the heart. In these cases the pericardium is involved from the myocardium. Dehio describes two forms of syphilitic affection, a gummatous pericarditis and a simple chronic inflammation. Lancereaux found a gumma of the size of a cherry stone in the parietal layer of the pericardium, along with gummata of the lungs.

Myocarditis gummatosa attacks by preference the wall of the left ventricle, and develops at the same time chronic peri- and endomyocarditis. These gummata remain a long time encapsulated without symptoms.

Syphilitic affection of the blood-vessels does not spare the coronary arteries, and certain cases of thrombotic occlusions with transudation in the pericardium have been attributed to syphilis. Schrötter quotes from Wanitschke a case of hereditary syphilis in a new-born child, in which the pericardium was involved in a large tumor situated in the upper lobe of the left lung. After agglutination and perforation of the parietal layer, pericarditis set in with sero-fibrinous exudation. The nature of the pericarditis was determined by the evidence of syphilis elsewhere.

In all cases the diagnosis rests upon the existence of other signs of the disease, or, when the condition is suspected, upon the results of treatment. It is needless to say that any case of suspected syphilitic origin imperatively demands the persistent use of an antisyphilitic treatment, especially of mercury by inunction, until the possibility of this infection is excluded by failure to secure results.

Pericarditis.

Pericarditis (*περί*, around; *καρδία*, heart), inflammation of the pericardium in the course of or as a sequel to some infection; or inflammation by extension of disease from some contiguous viscus.

History.—The anatomical lesions of the pericardium could not escape the notice of the earlier anatomists. Galen certainly saw the disease in animals and suspected the existence of it in man. Rondelet referred to it under the symptoms of pain, dyspnœa, and syncope. Riolan recognized the danger of the disease and suggested withdrawal of the effusion for the relief of pressure. Morgagni declared that the day was distant when the ability to recognize the disease would justify a paracentesis. Vieussens described the frequent occurrence of the concretio pericardii, that is, the adhesion of the two layers of the pericardium. Auenbrugger first noticed the bulging of the precordium and discovered the dulness on percussion. This discovery, however, excited no attention until it was utilized and published by Corvisart (1811) as a distinct means of recognizing the disease. Sénac described pericarditis in his work on the diseases of the heart. The indefiniteness of knowledge concerning pericarditis at this time may not be better shown than by the fact that Laennec (1819) doubted the possibility of making a correct diagnosis. It is a matter of history that Laennec had really heard the friction sound, but with all

his acumen he seems to have failed to appreciate the significance of it. So it was reserved for his *chef de clinique*, Collin (1824) to recognize the value of it and thus to definitely establish the diagnosis of the disease. Paracentesis pericardii was first done by Romero of Barcelona in 1819.

The later contributions of our century by Netter, Weichselbaum, and Banti have illuminated the subject chiefly from the standpoint of etiology, demonstrating the causative relation especially of rheumatism, tuberculosis, and pneumonia.

General Remarks.—Up to the close of the last century pericarditis was considered a rare disease. More precise knowledge of the nature of the affection and easier means of recognition prove it to be of quite frequent occurrence. Statistics upon this point differ, however, as they are derived from the diagnosis in life or from the results of post-mortem examinations. It is certainly true that many cases are recognized only upon autopsy, and still more frequently the fact that a previous existence of it is established by the discovery of lesions in the same way. These lesions, as will be seen, are sometimes so obtrusive as to establish the character of the disease at a glance. In other cases the relics of inflammation are of more difficult recognition or interpretation. Thus the pericardium is often found studded with white spots, the so-called tendinous or milk spots, the true relation of which it is necessary to know. These milk spots have been variously regarded by the anatomists. They appear upon both layers of the pericardium, but especially on the visceral layer of the right heart, and consist of cicatricial connective tissue covered with endothelium. From their situation in regions of the heart uncovered by the lungs they are supposed to be hyperplasias from friction. It is a fact that thickenings of the same character are found in other serous membranes, especially in the peritoneum in the region of the spleen. The milk spots are, therefore, to be regarded as friction scleroses and not as indication of inflammation. It is observed also that milk spots are more frequent in old age, a period in which pericarditis itself is rare. Therefore the statement of Duchek, who claims to have found evidence of pericarditis in over fifteen per cent. of autopsies, must be regarded as an over-estimate, for the reason that this author regarded milk spots as evidence of pre-existent pericarditis. The proportion given by Williglk, who found pericarditis in four per cent. of autopsies, is generally accepted, and all the more willingly because the observations were made during the earlier periods of life.

Pericarditis may occur at any period of life, but is most frequent in youth and early maturity, less so at the extremes of life.

Cnopf maintains, however, that pericarditis is not so rare in childhood as is commonly believed. Cnopf saw pericarditis ten times in 459 cases of disease of children. The ten cases were pretty uniformly distributed through the different ages from one to eleven years. The primary cause in these cases was either pneumonia or tuberculosis of the lungs; in two cases there was scarlatina.

The question as to the relative frequency of pericarditis and endocarditis as separate affections is difficult to determine. Hospital statistics differ in different years. Sibson's record shows three times as many cases of endocarditis. Probably this ratio would be accepted by most clinicians, but hospital statistics sometimes show a preponderance of pericarditis. This ratio may be an accident of the year, or it may be that endocarditis only seems more frequent because it leaves valvular lesions. Patients recover from or succumb to pericarditis as a rule more quickly. Cases do not accumulate as in endocarditis.

Etiology.

Pericarditis is said to be primary and secondary. By primary pericarditis it was intended to convey the idea that the disease might spring up spontaneously, for instance under the influence of "cold." This idea is now entirely abandoned. But the term primary is reserved for cases due to trauma, and to cases apparently dissociated from disease in any other part of the body. Puncturing wounds may certainly produce pericarditis, but the inflammation which results from a clean incision is not a true pericarditis. Aseptic instruments do not produce the disease, and true pericarditis results from trauma only when some infection is introduced by the intrument.

Dziembowski extracted a needle from the pericardium of a boy aged fifteen. Improvement followed without any complication and the murmur connected with the heart's action, which could be heard nearly twelve inches from the heart, immediately disappeared.

Contusions act in the same way. Micro-organisms are wont to colonize in the quiet regions offered by extravasated blood. The experiments of Schuller are interesting in this regard. This observer found that when an animal was inoculated with tubercle bacilli and the joints were subsequently injured, colonization of the bacilli occurred in the joints. An illustration of this kind of traumatic pericarditis was reported by Ulrich Lüchinger in the case of a farm laborer who fell from a hay wagon and suffered a contusion of the left side of the thorax, with simple fracture of the left leg. The injury to the chest was apparently trivial. There was no sign of fracture of the ribs or sternum. During the course of the third night

after the accident, the patient began to complain of palpitation of the heart and difficulty of respiration. Examination revealed pericardial friction over the right ventricle. Evidence of pericardial effusion was apparent in a few days and pleurisy with exudation followed a few days later (Whittier and Green).

Indirect trauma, as by penetration of foreign bodies from the œsophagus or stomach, acts in the same way. Here also may be included the occasional cases which result by contiguity from abscess of the spleen, from empyema, echinococcus of the liver, aneurysm of the aorta, caries of the vertebræ, affections of the mediastinum, inflammations of the mammary gland, extensive disease of the skin, etc. These cases are, however, not so likely to produce a simple inflammation as they are to introduce the organisms of infection from the outside air and thus produce a purulent or ichorous disease. All these cases are rare; they are little more than curiosities, or, as in the case of penetrating wounds from without, have more special interest for the surgeon.

But there are occasional cases in which pericarditis exists alone, that is, in which the disease is primary. These are cases which have been attributed to "taking cold." The process of taking cold is understood in our day as a determination of a cause of the disease to some particular organ. The cause which is thus determined exists in the body elsewhere, but sometimes in latent form. The process of taking cold may make a latent cause manifest.

Banti, Vanni, Mio, Belfanti, showed that infection with pneumococci or typhoid bacilli would produce pericarditis in animals if the pericardium had been subjected previously to chemical or thermic irritation. Rubino carried these experiments further and produced pericarditis by trauma, by blows over the pericardium, or by the application of ice. The experiments succeeded in both cases. Pyogenic micro-organisms were used with the production of suppurative pericarditis in the case of the blow, and in the case of trauma with the production of first myocarditis and secondarily pericarditis.

Thus it is known that the micro-organisms of diphtheria or of pneumonia may exist in the throat without evidence of disease, but exposure to cold may disseminate the micro-organisms or develop toxins and thus give rise to the signs and lesions of the disease. In the same way the diplococcus of pneumonia sometimes operates to produce meningitis in the absence of pneumonia. Ménétrier and Pineau reported a case of purulent pericarditis produced by pneumococci, as verified by culture and inoculation. There had been no pneumonia in the case. Foureur once found the Streptococcus pyogenes in the pus of a purulent pericarditis, but was not able to

find a dépôt of suppuration anywhere else in the body. So the tubercle bacillus has been found only in the pericardium, but of course always from some cryptogenetic source.

Virchow reported an illustrative case of isolated tuberculous pericarditis. On section of the much thickened pericardial wall an immense layer of tubercles could be seen next to the muscular tissue. The tubercles were full of giant cells but contained comparatively few tubercle bacilli. The case was regarded, like others previously observed, as one of protracted latent pericarditis going on to the production of highly vascular new connective tissue. Hemorrhage arose from the newly formed deeper layers. The first case that came under his notice was that of an old man aged eighty, in whom there could be discovered no other trace of tuberculosis. In some of these cases the hemorrhage is so great as to suggest at first a rupture of the heart.

The true disease, pericarditis, which is of interest to the clinician from its frequency and gravity, is always a secondary affection; that is, the pericarditis occurs in the course of some infectious disease. As a rule, the original disease is patent. It may be still in progress or will have only recently subsided, but sometimes the connection is more difficult to establish. The original affection may have long since disappeared and left little or no other trace, or the original malady may have been so trivial—a tonsillitis for instance—as to have escaped notice. Such cases are said to be of cryptogenetic origin. The knowledge of the nature of the disease diminishes the number of the cases of so-called primary pericarditis every year. Duchek saw it only once in 89 cases, Bamberger but four times in 63 cases, and Friedreich, with his wide experience, met but two cases in which he could discover no cause for the disease.

Cases of primary pericarditis were reported by Virchow, Kummel, Cornil. But it is impossible to eliminate the infections in these cases. Thus in the great majority of cases tuberculous pericarditis is secondary to tuberculosis of the lungs or pleura or to tuberculosis of the mediastinal or tracheo-bronchial glands. Sometimes the tuberculosis is latent. The fact is, that cases of primary tuberculous pericarditis become more infrequent every year, and most of the so-called primary cases are really consecutive to tuberculosis of the glandular system. Sometimes the source is secluded. For instance, Weigert has shown that in many cases the small glands situated in front of the anterior layer of the pericardium may be involved, and may be even in a state of caseous degeneration and yet easily pass unnoticed. Eichhorst reported cases of tuberculous ulceration of the pericardium consecutive to tuberculous ulceration of the

intestine. The streptococcus and the pneumococcus have been found, as stated, in the effusion of pericarditis, and not elsewhere.

Since the first demonstration by Pitcairn (1788), it has been recognized that rheumatism stands in the most intimate connection with pericarditis. The liability to heart disease as a complication of rheumatism is universally acknowledged, but attention has been hitherto more especially directed to endocarditis. The knowledge of the fact that the pericardium may be also involved is of more recent date. The proportion of cases in which the pericardium is involved is variously given by different authors. Bouillaud believed that the heart was affected in some way in every case of acute articular rheumatism. Williams thinks that in every 100 cases 75 are affected. Leudet puts the ratio at 22 to 100, Ball and Sibson at 20 to 100, Wunderlich at 19 to 100, Duchek at 16 to 100. Bamberger claims that 30 per cent., Chambers and Thompson 16 and 20 per cent. of cases arise from rheumatism. But regard is here had chiefly to endocarditis, and pericarditis is scarcely considered. On the other hand, Latham found pericarditis only 7 times in 136 cases of rheumatism. When pericarditis occurs in rheumatism it shows itself by preference between the fourth and fourteenth days of the disease. The disease is certainly more frequent in the severe cases and is especially liable to occur when the inflammation wanders rapidly from joint to joint. Pericarditis is also much more frequent in the young. The complication is much less liable to ensue when the disease is oligarticular or monarticular, and in cases of subacute and chronic rheumatism. Thus Fuller found pericarditis only twice in 44 cases of subacute rheumatism, and most authors deny the complication altogether in chronic rheumatism. Yet it does sometimes occur. Romberg, Walshe, Trastour, Ball, Cornil, Charcot report cases.

Sometimes the pericarditis precedes the rheumatism; Stokes, Graves, Taylor, West reported cases of this kind. Halle collected cases in which the pericarditis occurred 22 times in 27 cases of "concealed" rheumatism. It was for a long time taught that pericarditis never occurred in gonorrhœal rheumatism, but Ricord, Raynaud, Lehmann, Basnier reported cases with this complication, and Leyden and Councilmann observed the distinct lesions, and verified the cause by demonstrating the presence of the gonococcus in the pericardium. The fact that the inflammation of the membranes of the heart sometimes precedes the affection of the joints shows that pericarditis is produced by the same cause as the rheumatism. The fact is that pericarditis may occur in the course of any disease that is caused by the invasion of micro-organisms. Rheumatism is the

most frequent of any one cause of pericarditis, partly because of the frequency of rheumatism. Rheumatism is one of the most frequent of a great group of diseases produced by micro-organisms. "Rheumatism is a disease which turns its microbes now into the pleura, again into the joints, now into the endocardium, again into the pericardium" (Fraentzel).

It is known that the pericardium is not alike affected by all micro-organisms. Certain of them show distinct predilection for this structure, and so notoriously is this true of acute polyarthritis that the possibility of pericarditis is sometimes excluded in the absence of a history of rheumatism. The relationship of the two affections is further shown in the connection with chorea, which is recognized as a kind of cousin germane with rheumatism. In 71 cases of chorea Roger found pericarditis 5 times, endopericarditis 19 times, endocarditis alone 47 times. Ollivier found 12 cardiopathies in 30 cases, pericarditis once, endocarditis 11 times.

The next most frequent cause is tuberculosis. This affects the pericardium in both ways. That is, the process may infect by contiguity, or a vomica may open up the pericardium to produce the disease in a mechanical way. Or the micro-organisms of tuberculosis may lodge and multiply upon the serous surface just as upon the cerebral meninges or tunica vaginalis as conveyed thither in the lymph and blood supply. Bamberger's statistics show pericarditis in fourteen per cent. of cases of pulmonary phthisis, but this is a decided under-estimate because it was made at a time when pleurisy, which is excluded in this consideration, was believed to be a separate disease.

Pleurisy itself is frequently attended or followed by pericarditis; indeed, the second place in causation was formerly assigned to pleurisy. In our day pleurisy is regarded as an expression mainly of tuberculosis, but the fact is that both the pleurisy and pericarditis result from the same cause, though the pleura is naturally, as a rule, affected first. But cases of pleurisy from other cause, as from the action of the diplococcus of pneumonia or from pyogenic micro-organisms, may produce pericarditis.

The next most frequent cause is affection of the lining membrane of the heart. In fact, the association of endocarditis and pericarditis is so frequent as to make a differential diagnosis at times difficult, and it is often impossible to declare the order of precedence. The intimacy of this relationship is indicated by the term endopericarditis.

Next in order comes pneumonia. The fact that the diplococcus of pneumonia produces pericarditis has become so well established as to have led to the attempt to declare the character of the prognosis

by the discovery of the diplococcus in the effusion. Leudet found pericarditis six times in eighty-three autopsies of pneumonia. This ratio may also be considered an under-estimate for the reason that, as stated, the diplococcus sometimes produces pericarditis without pneumonia. Fraenkel reported cases of this kind. Weichselbaum maintains that the pericarditis which occurs with pneumonia is produced by the diplococcus of pneumonia, and the pericardium is to be looked upon as a rarer localization of the same cause. Pericarditis is, he declares, more frequent in pneumonia than is commonly believed, as he found that he could in proper culture develop the Diplococcus pneumoniæ in cases where the pericardial fluid was perfectly clear. But in one case of pleuro-pneumonia he found the diplococcus in the liquor pericardii, so that everything was ready for an outbreak though the pericarditis did not occur. It is observed that pericarditis may develop at any period in the history of pneumonia. Broncho-pneumonia, especially that form, the so-called cellular pneumonia (Finkler), produced by influenza, is less frequently followed by pericarditis.

The etiological relation of scarlet fever was observed by Kruckenburg as long ago as 1820, and afterward by Bouillaud and Trousseau. Thore devoted himself to the study of scarlatinous hydropericarditis. The complication develops usually between the fifteenth and thirtieth day (Petit).

Variola is rarely followed by pericarditis, though cases were reported by Andral, Gintrac, Huchard, and others. Pericarditis after measles was observed by Frank, Barthez and Rilliet, Dufour; after erysipelas by Jaccoud, Duroziez, and Zülzer, who considers the complication more frequent than is generally believed. Denucé actually found the streptococcus of erysipelas with distinctive characteristics in two cases of pericarditis which followed erysipelas of the face. Diphtheria is a most infrequent cause, so much so that Sanné doubted the relationship even in the face of a pericarditis. Authors differ with relation to typhoid fever. Mussy believed it frequent, Homolle considers it rare. Petitfour collected six cases. The precedence of typhoid fever, diphtheria, scarlet fever, speaks much more decidedly for myocarditis than for pericarditis.

Pyæmia is a frequent cause. It is believed by many authors that most cases of pericarditis are due to secondary invasions by the micro-organisms of pus and that the various infections act only as pioneers in breaking down the barriers for subsequent invasion. Rheumatism itself is thought to be due to the invasion of micro-organisms closely allied if not identical with those of pus. The frequent repetition of attacks is explained by the liberation of these

micro-organisms from secreted depots. It has long been known that diseases attended with suppuration may be followed by pericarditis. Tuberculosis, especially of the lungs, is always a mixed infection in the course of time. It is said of scarlatina that only those cases which show affection of the joints are followed by pericarditis, and the affection of the joints has long been recognized to be an expression of pyæmia. According to Kolisko, the pericarditis and the joint affections are due to the invasion of the streptococcus which makes its inroads from the throat, that is, from the angina of scarlatina.

Kirke, Willigk found pericarditis five times in ninety-one cases of purulent septicæmia. In one case reported by Homolle, puerperal pericarditis was found in the new-born child of a mother who had died of pyæmia. In all of the thirty-six cases reported by Bednar, the disease in the new-born child could be attributed to puerperal processes in the mother. Before the days of asepsis, pericarditis was a frequent complication of surgery. Weber once found purulent pericarditis in a case of inflammation of the umbilical cord.

The connection with scurvy has been at times so pronounced as to make pericarditis epidemic, though this complication of scurvy is less frequent than pleurisy. But hemorrhagic pericarditis from scurvy sometimes assumes epidemic proportions in the north of Russia. Koch declares that the pericarditis of scurvy was the morbus cardiacus of the ancients, an exceedingly grave affection, whose gravity was due to the fact that the inflammation of the membrane was attended with degeneration of the heart muscle.

Pericarditis supervenes also in other diseases attended with the same degradation or degeneration of the blood, such as hæmophilia, the hemorrhagic diathesis, purpura, morbus maculosis, leucæmia, diabetes, cirrhosis of the liver, etc. The pericarditis observed in cancer and sarcoma may result in this way or may be a direct development of the growths by metastasis. Syphilitic pericarditis has been seen in the new-born.

Pericarditis is not at all infrequent in Bright's disease. Taylor found it in one-third of the cases. Bamberger more correctly fixed the ratio at fourteen per cent. It is certain that the pericardium is less frequently affected in this disease than is any other serous membrane. The authors differ as to the relation of the pericarditis to the particular form of nephritis. Grainger Stewart and Dickinson found it more frequent in parenchymatous nephritis; Raynaud, Lécorché, Talamon in interstitial nephritis. Mercklen found pericarditis rare in acute nephritis, more frequent in the chronic form, and espe-

cially common in the interstitial form which was accompanied with hypertrophy of the heart. The pericarditis shows itself usually at a later period in the disease, sometimes with uræmia, in what Keraval calls uræmic pericarditis. The inflammation is usually dry, but is sometimes accompanied with abundant fibrinous exudation. As a rule, there is neither fever nor pain. This pericarditis is usually a forerunner of death.

Pericarditis is not very rare in cholera, and is quite common in dysentery, along with or independent of rheumatism, which sometimes follows dysentery. In erysipelas, diphtheria, and cerebro-spinal meningitis, pericarditis not infrequently constitutes the last link of the disease process.

But pericarditis may ensue upon even the lightest infections. Perhaps one of the most interesting statements that could be made in illustration of this fact is that of Bednar, who several times observed pericarditis supervene after vaccination. In one case an acute dermatitis developed in twenty-four hours after vaccination, with a simultaneous pericarditis. In a second case a sharp diarrhœa, subcutaneous abscesses, and pericarditis proved the order of sequence. In a third case the connection was not so clear; the pericarditis developed on the thirteenth day after vaccination, without intervening disease.

The pericarditis of drunkards is not to be ascribed to the direct action of alcohol, but to the liberation or localization of the diplococcus of pneumonia, tuberculosis, pyogenic micro-organisms, etc.

Pericarditis is more frequent in youth because of the greater frequency of the infectious diseases at this time. Liability occurs at the age of five and begins to cease in middle life, though exceptional cases have been reported at both extremes of life. Billiad, Homolle, Rauchfus, Steffen, Henoch all reported cases of intra-uterine infection. Letulle reported cases of death from purulent pericarditis on the seventh and thirteenth day after birth. Cox reported a case of pericarditis in an infant eleven days old, and Hawksley a case of extensive exudation of pus into the pericardium in an infant aged five months. Mejard contends, with the report of a case between seventy and eighty years, that acute pericarditis is not so very rare in old age. Willigk and Vulpian reported cases in advanced life. The disease is certainly more frequent in the male sex. According to Barthez and Rilliet the proportion is as great as 21 to 3. Bamberger makes it less, to wit, 38 men to 25 women; Sibson still less, 35 men to 28 women. Of these women fully two-thirds were domestics.

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Morbid Anatomy.

The infection of the pericardium may be circumscribed or diffuse. The natural secretion may be arrested or more frequently increased, sometimes to the utmost distention of the sac, or changed by admixture of pus, blood, etc. The disease may be arrested at the first stage, or may continue to the second with the effusion of fluid, or may result in adhesion of the visceral and parietal layers, sometimes with entire obliteration of the sac. When the disease is circumscribed, it is confined to the visceral layer at the base of the heart. In lighter localization it may be limited to the upper cul-de-sac, that is, to the extension of the visceral layer over the great vessels at the base. In all cases the visceral layer is the most affected.

Arrest or diminution in the natural secretion constitutes the form distinguished as pericarditis sicca. This form is recognized by the dryness of the serosa. The membrane loses its lustre, becomes cloudy, opaque, or is covered with arborescent vessels. The disease may undergo resolution at this stage and all the signs of pericarditis may disappear. Sometimes the pericardium is left rough or shaggy, in other cases apposed surfaces may adhere, to lead, as stated, to more or less complete obliteration of the pericardial sac. But the adhesions in a case of simple dry pericarditis are usually circumscribed. More frequently, however, the secretion is increased to constitute the pericardial effusion. The effusion may consist simply of an increase in the natural fluid and remain thus perfectly clear and serous. As a rule, however, the clear fluid is interspersed with floccules of fibrin, and the presence of these floccules distinguishes the effusion from the passive exudation which occurs in the course of a general anasarca, the so-called hydropericardium.

The fluid accumulates first in regions where it is determined by gravity, and this region will be determined by the posture of the patient. In a case of any severity, when the patient lies recumbent, the fluid collects at the base of the heart and for some time may escape recognition by percussion. In a lighter case with the person in the upright posture, or in a more severe case where the patient is unable to lie down, the fluid gravitates toward the region of the apex, and is recognized by dulness in the neighborhood of the ensiform cartilage. As the accumulation increases it fills up the pericardial sac, and finally distends it to produce a great globular tumor, which may take up a large part of the front and left chest.

The quantity of fluid usually ranges from six to twelve ounces (200 to 400 grams). It rarely exceeds one-half to one pint, but may

in extreme cases amount to a quart or more. Corvisart and Louis cited cases in the adult of 1,000 to 1,200 grams (two pounds) and more. Mortagne reported 1,850 grams, of which 1,600 were withdrawn by puncture. Gosselin once found two litres of liquid in a pericardium enormously extended (Petit).

Andral once found two pounds of blood in the pericardium, Alonzo Clark one gallon of sero-purulent fluid, Corvisart eight pounds of serum in the pericardium. Martin reported a case of pericarditis with an effusion of three and one-fourth pounds of purulent matter. But the greatest accumulations are found in the hemorrhagic effusions of scurvy, in which infection the effusion may amount to from two to six pounds. Kyber claims to have found three to ten litres of fluid in cases of scorbutic pericarditis. No other form of pericarditis furnishes such a quantity of fluid (Koch). In these cases the pericardium looks like a bluish, glistening bladder, which extends from the manubrium to the ensiform cartilage, displacing the left lung and making pressure upon the diaphragm.

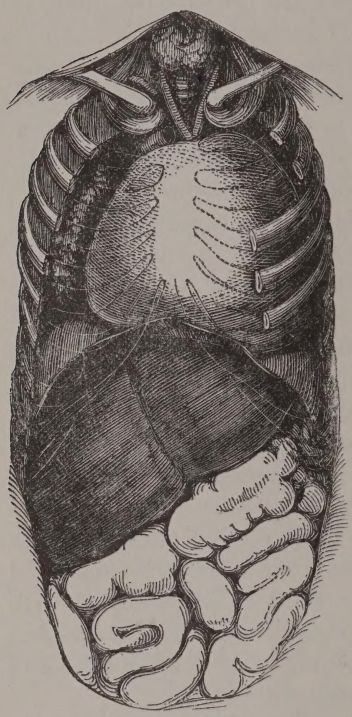

Fig. 1.—Pericardial Effusion with Displacement Downwards of the Liver.

The effused fluid is, as stated, usually serous or sero-fibrinous. It may subsequently become purulent. In fact, this is the usual history of pericarditis. In all cases, even in those in which the effusion is perfectly clear, pus corpuscles may be found in the bottom of the sac or in the fluid which is last withdrawn. But the fluid is said to be purulent when the pus corpuscles accumulate to such an extent as to make the fluid more or less opaque to the naked eye. The disease is then said to be a purulent pericarditis. Sometimes the transition is artificially produced by accidental infection, as in puncture with instruments not perfectly aseptic. Thus it has been observed that a fluid which was serous at first has become purulent upon successive punctures. Sometimes the fluid is purulent from the start. Foureur described a case in a woman, aged thirty-eight, in which the effusion was purulent from the start. Glaser and Mirabeau reported similar cases. Sometimes the pus undergoes a

subsequent transformation into ichor. This change is observed more especially after communication with the external air, as after perforation in ulcer of the stomach, cancer of the œsophagus, etc.

The effusion may also be hemorrhagic. The blood may be present only in small quantity, just enough to tinge the fluid, or the effusion may be more decidedly sanguinolent, or be composed largely or almost entirely of blood. Hemorrhagic effusions are observed more especially in tuberculosis, carcinoma, and in diseases in which the blood is degraded, scurvy, leucæmia, purpura, etc. Hemorrhagic pericarditis is seen also in black measles, variola nigra, virulent scarlatina, and in the course of any disease upon which is engrafted the hemorrhagic diathesis.

According to the statistics of Louis, in 39 cases the fluid was found serous 9 times, purulent 7 times, sero-sanguinolent 10 times, sero-purulent 13 times. But as a rule, serous effusions greatly predominate.

The pericardium itself shows various changes. Sometimes, as stated, the surface is simply hyperæmic. It loses the glistening lustre peculiar to healthy serous membranes and becomes more or less opaque, thick, and soft. Under the to-and-fro motions of apposing layers, sticky with inflammatory products, the surface becomes roughened like a Russian bath towel or presents the appearance so inimitably described by Laennec, of butter on the surface of plates which have been suddenly separated. The internal surface of the pericardium presents, therefore, the appearance of an ulcer. In fact, Rindfleisch compared it to a suppurating wound. Perforations may ensue so that the sac may be opened externally, or what is more frequently the case, may be connected with the lungs to constitute a pneumopericarditis or a pyopneumopericarditis. In other cases fistulous tracts may form and pus may be discharged at distant sites, as at the root of the neck, subcutaneously in the precordial region to form a fluctuating abscess, below the diaphragm to constitute one of the forms of subphrenic abscess, or by longer tracts to appear by the side of the vertebral column in the loins to resemble the sinking abscess of vertebral caries. Lavatier reported cases of perforation, Wyss of pericardial fistula.

Sometimes the surfaces are connected by filaments of fibrin to give the pericardium a shaggy appearance. This condition was recognized by the ancient authorities as the hairy hearts, which were supposed to be evidence of great bravery. Haller said that Leonidas, Lysander, and Aristomenes were thus distinguished by the presence of stringy fibrin on the heart.

Sometimes the pericardium is much thickened. Broadbent and

Horn reported cases of extraordinary thickness of the pericardium, Nunier a case in which the pericardium was covered by thick false membrane imbricated like the scales of a pineapple.

Sometimes the pericardium undergoes calcification. This process is more commonly a deposit in or transformation of a purulent effusion.

Fig. 2.—Cor Villosum.

Cases of such calcification of the pericardium were recorded by Tiessier and Richards. Drummond reported a case of extensive calcification of the heart in a sailor aged forty-three, who was able to perform his work up to a few weeks before his death. On autopsy it was found that the pericardial sac was nearly obliterated and the pericardial layers were extensively calcified. The process of calcification had extended so as to involve the heart muscle, in which it had developed great bone-like plates which had to be sawn through and which reached a thickness in spots of one inch. The whole posterior surface of the right ventricle was composed of a triangular chalk plate three inches high, three and one-half inches broad at the base. A thick bone-like mass ran across the whole left ventricle, penetrating the entire wall of the heart like a wedge and reaching into the cavity of the left ventricle.

Variot reported a case in which the left ventricle was covered by a solid, hard plate of chalky matter on its whole anterior face. The plate extended to the posterior aspect of the ventricle and reached down to the diaphragm. The apex of the heart was free. Microscopic investigation showed that the chalky mass was deposited between the still recognizable layers of the pericardium. The curious fact about the case was that this extreme alteration was latent up to a period shortly before death, when the patient was affected with pleurisy and recalled a former attack of heart disease.

But the changes are not limited to the pericardium. They extend so as to involve the heart muscle itself. Virchow first called attention to the extensive involvement of the heart muscle in pericarditis, and that, too, not only in the chronic but in the quite acute cases. Surfaces of greater or less extent or the whole heart muscle may undergo fatty degeneration. Purulent infiltration may occur in the upper layers and soon lead to degeneration and dilatation, but from

earlier stages the muscle may entirely recover. The gravest changes are found in scurvy in which the heart suffers extreme degenerative changes, that is, hemorrhages occur in the heart muscle, in the endocardium, and in the valves. The muscle loses its fresh red color, appears yellowish-brown or violet, becomes fragile or fatty, sometimes shrunken to two-thirds its thickness, dilated, and thinned. There may be nearly always observed in these cases an accumulation of fat about the base of the heart under the serous membrane, and the accumulation is at the cost of the muscle substance, with which it is intimately united (Sansom-Himmelstiern).

Symptoms.

Pericarditis sometimes runs its whole course without symptoms. Sometimes the pericarditis is overshadowed by affection of other parts of the heart, as of the myocardium or endocardium. Light cases are especially liable to be overlooked. This fact has given rise to the view that pericarditis is a rare affection. Later observations have confirmed the view of Bauer to the effect that "formerly and up to the close of the last century, pericarditis was considered a rare disease; since then it has been proved to be of quite frequent occurrence." The statement made once by the author of this paper, at the Cincinnati Academy of Medicine, that pericarditis is ordinarily oftener overlooked than recognized, which was denied by several members, was strikingly confirmed by an ex-interne of one of our largest hospitals, who observed that of the five cases which had occurred in his service, the diagnosis was not once made *intra vitam;* the existence of the disease in every case was made apparent only on the post-mortem table. Letulle declared long ago that of all acute or chronic diseases, pericarditis oftenest runs a latent course.

The failure to recognize pericarditis depends upon the fact that so few of the symptoms of the disease are local. It is only in the face of effusion, and mostly of some magnitude, that signs pertaining to the heart are manifest. Then, medical teachers and clinicians so often emphasize the fact that pericarditis is a sequel to rheumatism that in the absence of rheumatism the existence of pericarditis is not even suspected. The failure to recognize pericarditis depends largely upon the fact that the existence of the disease is not suspected; for when the suspicions of the practitioner are aroused, the diagnosis is generally easy. A striking illustration of this fact the writer saw a few years ago. The patient had been the victim of unsuspected pericarditis with effusion for fifteen years, at least the same symptoms had been present in greater or less degree all that

time. The prominent symptoms were pallor, dyspnœa, such vertigo as to compel the recumbent posture, occasional cough, and a pulse so feeble as to fade away when the arm was held at right angles to the body. Digitalis and alcohol administered from time to time had obviated an imminent collapse. There was undoubted increase of dulness over the region of the heart which had been taken for dilatation. By aspiration one pound of serum, at first clear, later brownish and flocculent, was withdrawn from the pericardium. The symptoms of immediate danger soon disappeared. But the long pressure or perhaps the incident myocarditis had weakened the walls of the heart to such an extent as to prevent perfect recovery. This was the third case under the observation of the writer of long-continued unsuspected pericarditis with effusion. In every instance inquiry had been made as to the previous existence of rheumatism, and in the absence of it no further attention had been given to the heart. It is not safe to exclude pericarditis in the absence of pre-existent rheumatism, first because pericarditis arises in consequence of so many other conditions, and second because the pericardium may be in rheumatism the first and only joint (so-called) affected. Sometimes large effusions accumulate insidiously and sometimes even suddenly. Thus Baumgärtner reported a case of fatal acute effusion of fluid into the pericardium without any previous symptoms, and Blake a case of sudden pericardial effusion.

Pain is a pronounced symptom of pericarditis and is more frequently present than in affection of the myocardium or endocardium. But the pain may vary in every degree of intensity. Sometimes it is so slight as to be overlooked or to be considered as a mere pleurodynia or intercostal neuralgia. Sometimes, exceptionally, it is as intense as an angina, but in the worst cases it is rarely as severe as the pain of pleurisy. Pain is often influenced by change of posture, or may be elicited or increased by pressure, as by examination with the stethoscope.

Palpitation is frequent, but is usually more or less transitory. Sometimes the palpitation depends upon the associate myocarditis. Palpitation may be so extreme as to amount to a delirium cordis, but usually only in the last stages of a bad case, and for the most part then as a result of dilatation from weakening of the myocardium. But a peculiar feeling of uneasiness and distress is more common and more persistent. This sensation, which is usually described as a precordial anxiety, distinguishes the onset of most cases. The patient may declare that he feels no pain, while the countenance expresses anxiety, which the patient will locate in a vague and indefinite way in the left side of the chest.

Dyspnœa shows itself in pronounced cases. Here, too, the interference with respiration is more a vague and general distress than the result of a distinct pain. The act of inspiration is not cut short as in pleurisy, but is more hurried and superficial.

The posture is peculiar. Most cases observe a dorsal decubitus in the semi-recumbent posture. The head and shoulders are elevated so that any fluid present may gravitate to the bottom of the sac and thus relieve from pressure the great vessels at the base.

The pulse is not necessarily affected. It is remarkable how the pulse may sustain its volume and tone in the presence of considerable exudation. As in all cases of interference with the discharge of the venous blood, distention of the veins of the neck is sometimes seen in pericarditis. Undulation of the veins is of frequent occurrence. Inspiratory swelling of the cervical veins is seen sometimes in great exudations. Sometimes there is ectasia of the veins of the chest-wall.

Fever plays no rôle in pericarditis. Sometimes it is absent altogether; when present it is highly irregular. Pyæmic processes show chills and fever connected with the pyæmia. The temperature in tuberculous pericarditis is sometimes subnormal.

The urine is usually high-colored and shows all the characteristics of the urine of stasis, that is, it may contain more or less albumin, or shows red blood corpuscles, sometimes, though rarely, hyaline casts.

Rarer symptoms are hoarseness and aphonia from implication of the left recurrent or laryngeal nerve; attacks of syncope from anæmia of the brain, the result either of pressure upon the great vessels or of heart failure from myocarditis; singultus and vomiting, the result of pressure upon the phrenic nerve; somnolence and stupor, delirium, convulsions, and coma from hyperæmia, the result of pressure upon the vena cava or of the accumulation of toxins in heart failure. Austin Flint recorded cases of pericarditis marked by delirium, Abbott a case of attempted suicide during the delirium of pericarditis. These signs, or some of them, are wont to show themselves in the last stages.

Thus the subjective signs may be vague and indistinct, or may be obvious and obtrusive, and it may be repeated that scarcely any disease varies more in the severity of its symptoms. But, fortunately for the diagnostician, the physical signs are much more sure, so that the disease is overlooked, as stated, only because it is unsuspected and physical examination is not made.

Physical Signs.—Inspection may show the disturbed action of the heart in palpitation, sometimes in the first stage, in increase in the

area of impact. Where effusion has taken place, the heart may be pushed away from the wall of the thorax so that even the apex stroke may not be visible. Weakening and absence of the apex stroke occurs, as a rule, in pericarditis independent of the presence of fluid in consequence of the coincident weakening of the myocardium. The presence of a good stroke in the radial pulse, while the apex stroke is weak or absent on account of the accumulation of fluid, is a much more characteristic sign of a pure pericarditis. Another valuable sign is the change in the position and strength of the stroke with change of posture. Thus the apex stroke, invisible or impalpable in the recumbent posture, may become visible or palpable when the patient lies upon the side or adopts the knee-elbow posture.

Sometimes the whole precordial region is protruded. This so-called vaulting or *voussure* of the chest may be seen only when the chest-walls are resilient, more frequently therefore in childhood and early adolescence. It cannot occur in the adult with effusion of less than 400 grams (over twelve ounces). According to Louis and Woillez it is nearly always present with effusions of 500 grams, but Potain saw it present in much slighter effusions and regards it, therefore, as a sign of but mediocre value, as an index of quantity.

The *voussure* is so rare as to be almost never seen in later life, as even great accumulations find place in the cavity of the chest under the easy retraction of the lungs. In fact, the light degree of protrusion which shows itself in obliteration of the intercostal spaces is to be accounted for rather by implication (paresis) of the intercostal muscles themselves by contiguity of structure. Pressure upon the diaphragm may protrude the epigastrium, but these protrusions are also rare for the reason, as stated, that the lung so easily recedes under any kind of pressure.

Percussion shows dulness corresponding to the condition and extent of the effusion. As the fluid usually accumulates first in the complementary spaces, it may be recognized in the second or in the fifth intercostal space near the sternum. Dulness at the second intercostal space may belong to the heart itself, but dulness in the fifth intercostal space is more directly due to effusion in the pericardium. Dulness to the left beyond the apex stroke is a sign of unmistakable value. Whatever doubt may pertain to the diameters of the heart itself, the position of the apex may be actually determined by inspection or palpation; and dulness to the left, or lower down than the apex stroke, would point pretty clearly to the presence of fluid. It will be remembered that the heart itself is a triangle whose base is upward and whose apex is downward and to the left, while the pericardium is a loose sac attached only to the vessels at the base. As

the sac fills under distention it assumes the form of a truncated cone, the base of which is below and the top above the heart. Under great distention the outlines of the pericardium may be distinctly developed. The diagnosis is usually easy in these cases under the general dulness to percussion over the region of the heart. Anything like an absolute dulness of wide extent that reaches up as high as the second rib should excite the suspicion at once, so far as heart disease is concerned, of the existence of pericarditis with effusion. But the diagnosis may be reached by the recognition of comparatively small quantities of fluid in certain definite regions.

The pericardium has two recesses or sinuses which may be looked upon as complementary spaces. The upper of these two spaces is situated at the base of the heart where the outside layer extends above the origin of the great vessels, reaching its highest point on the aorta, while the inside layer is attached to the auricles. The second recess or sinus is much smaller, but is of much more important diagnostic value, as it is situated at the right lower border of the pericardium corresponding to the sternal angle of the fifth right intercostal space (Ferber).

Rotch found that when he injected the pericardial sac with melted cocoa butter, the first increase of dulness was to be found at the bottom of the sac to the right of the sternum, and remarked that flatness two to three centimetres from the edge of the sternum in the fifth intercostal space is almost sufficient alone to mark the presence of effusion in the pericardium. Ebstein supports this view that the fluid accumulates first in the lower part of the sac, and that percussion recognizes dulness first at the sternal end of the fifth intercostal space at the so-called heart-liver angle.

As to the quantity of fluid that may make itself manifest to percussion, it may be remembered that Luschka has shown that when the heart is moderately full of blood the pericardium may take up 180 grams (6 ounces) of water without any forcible distention. Sibson found that the pericardium of an adult male would hold 14 to 22 ounces of water, while that of a boy aged six to nine years contained but about 6 ounces. It is admitted that quantities of fluid less than 4 ounces in the adult may scarcely be recognized by percussion.

Gerhardt called attention to the fact that the dulness observed in the recumbent posture in pericarditis is much increased in standing or sitting. The change of posture enlarges the area of dulness under pressure of the fluid by gravity as much as one-third to one-half, and sometimes bulges the intercostal spaces.

The evidence furnished by auscultation is of great value, though it is not so decisive as that of percussion. Moreover, it does not last

so long. The most important sign thus obtained is the friction sound, which results, as the name implies, from the apposition of the surfaces of the pericardium roughened by the products of inflammation. The friction sound was first heard by Laennec, who likened it to the sound of a new saddle under the cavalier. Laennec made many acute observations concerning pericarditis, especially in the field of pathology, but seems to have attached little importance to the friction sound, the full significance of which was only established by Collin in 1824. The friction sound corresponds in a general way to the movements of the heart. It is usually heard both with the systole and with the diastole, whence it has received a simple but expressive Anglo-Saxon designation as a "to-and-fro" sound. The sound is often triplicate, has a presystolic, a systolic, and a diastolic epoch. Sometimes the sound is quadruplicate in periods which correspond to the systole of the ventricle and the diastole of the auricle, and to the diastole of the ventricle and the systole of the auricle. While it corresponds in a general way to the systole and diastole, it is not so distinctly synchronous with the movements of the heart as are the valve sounds. The friction sound is usually heard first at the base, and may afterward become more general. The character of the sound is altered, or the sound may disappear with change of posture. Thus a sound which may be distinctly audible when the patient lies on his back may disappear entirely when the patient lies upon the right or left side, as inflamed surfaces may be removed from contact with each other. The sound varies in every degree of intensity, from the crumbling of tissue paper to the creaking of leather. Sometimes it resembles the crunching of snow under the feet; sometimes the sound is more distinctly rasping or grating in character; sometimes it is so rough that it may be felt as a fremitus under the fingers.

Digby reported a case of pericardial friction sound so loud that it could be heard a distance of nine feet. The sound was loudest at the apex. It was systolic and had the character of creaking leather. The sound stopped every fifth or sixth beat, and after three or four beats returned. This phenomenon was heard in the case of a girl aged eleven years who had suffered two and one-half years previously with scarlet fever, and the murmur had been heard, according to the parents, for three years without the complaint of the least pain or palpitation after effort. Unfortunately the details of this curious case are so meagre as to leave the pericardial origin of the sound somewhat in doubt. Smith reported a case of pericardial sound with musical murmur.

Change in the posture of the patient may produce variations in

the character or intensity of the sound. Pressure, as with the stethoscope, may intensify a sound feeble before, may change the character of it, or may develop a sound unheard before. Exercise of the body may have the same effect. The duration of the friction sound is very variable—sometimes it is present but a few hours; sometimes it may be heard during a greater part of the course of the disease; sometimes it disappears to reappear in the later course of the disease. Schrötter speaks of a case in which the friction sound was present in great distinctness for four months.

The friction sound disappears:

(1) Under resolution in which process the roughness disappears by absorption;

(2) Under adhesion, which agglutinates the apposed surfaces;

(3) Under effusion, which separates them.

With the absorption or discharge of the effusion roughened surfaces may again come into contact and renew the sound. The pericardial friction sound does not disappear so quickly under slight effusion as in the case of the pleura, for the reason that the more powerful contraction of the heart forces the surfaces into more abrupt contact.

Auscultation may appreciate also alteration in the sounds of the heart itself. Ordinarily in average cases the sounds of the heart remain unaffected and may be distinguished as pure sounds in the absence of friction sounds, or when the friction sounds are not distinctly synchronous. The heart sounds become weakened under effusion; under great effusion they may be absolutely inaudible. This muffling or weakening of the heart sounds correspond to the diminution in impact of the heart. When the apex stroke cannot be seen or felt the heart sounds become feeble or inaudible. But the weakening of the heart sound is not due so much to the accumulation of fluid as to the weakening of the muscle of the heart itself. The pericardium is a distensible sac and the force of the heart is so great as to overcome outside pressure so long as the muscle tissue remains sound. Schrötter comments here upon the fact that the heart sounds of the fœtus are heard through a much greater quantity of amniotic fluid than ever occurs in a pericarditic exudation. Hence it is, as stated, that there is no pulse peculiar to pericarditis, and the weak pulse is an indication rather of myocarditis than pericarditis.

Auscultation further reveals at times galop rhythm, which Petit regards as "a precious element in diagnosis in the beginning of the disease." Potain attributes the galop sound to loss of tone in the myocardium so that the blood falls into the ventricle without obstacle

up to the moment of sudden complete distention. The sound of this sudden distention produces the presystolic shock, the two normal sounds constituting the remaining features of the galop. Schrötter attributes the sound to a duplication of the second sound, due to a saturation of the pericardial layers at the beginning of the exudation. The more sticky surfaces adhere at the moment of systole, and the tearing loose of the heart from the parietal pericardium at the moment of diastole gives rise to a short sound which is appended to the diastolic tone developed in the interior of the heart. The galop rhythm, in the opinion of the writer, is better explained as irregular contractions of the heart muscle, as described more fully in the study of myocarditis. However produced, the sound is of all the more value because it precedes even the friction sound.

Pins called attention to the bronchial respiration which is heard below the pericardial sac from compression of the lung by gravity. This sound may be made to disappear with change of posture, perhaps to reappear in other parts of the lung. The bronchial respiration, the dulness, and the pleuritic rub may all disappear in the knee-elbow posture. Finally Riess called attention to certain sounds in the stomach consonant with the heart sounds in adhesion of the pericardium.

Diagnosis.

The diagnosis rests upon the existence, or the history of the previous existence, of some infection, and is especially directed to the pericardium under the coexistence or pre-existence of rheumatism and tuberculosis. Any infectious process may produce pericarditis, but typhoid fever, diphtheria, and scarlet fever are much more likely to produce myocarditis. It will be remembered that pericarditis may occur after an affection as trivial as quinsy, and that the originating malady may have passed unnoticed or may have been forgotten. So pericarditis is often insidiously introduced. The chief signs upon which the diagnosis is based are pain, which is present, according to Sibson, in seven of ten cases. It is usually located in the præcordium, but is sometimes felt in the back between the shoulders under the left scapula, and very frequently in the epigastrium. It is usually pronounced and is exceptionally so severe as to simulate angina pectoris. Dyspnœa was observed by Mayne ten times in eleven cases. The dyspnœa is usually due to complications, myocarditis, endocarditis, and is sometimes accompanied by sighing respiration. Palpitation was observed by Hache fourteen times in twenty cases. Arhythmia is a frequent sign of pericarditis.

The diagnosis really rests, however, upon the physical signs and

especially in the first stage upon the friction sound, which is superficial and is usually most distinct at the base. The friction sound shows changes or disappears under changing posture, and is intensified or annulled by pressure.

The to-and-fro sound of pericarditis is differentiated from the friction sound of pleurisy by the fact that it corresponds with the movements of the heart and not with the acts of respiration. Arrest of respiration as by holding the breath, stops the friction sound of pleurisy, but has no effect upon the friction sound of pericarditis. Exceptionally, however, the movements of the heart may bring inflamed surfaces of the pleura into contact. In these cases the diagnosis may be reached only by consideration of the other signs of pericarditis.

As to the distinction between pericardial and pseudo-pericardial (pleuro-pericardial or extra-pericardial) symptoms it may be remembered that the sense of oppression predominates in pericarditis, and of pain in pleurisy. It is sometimes very difficult to distinguish a pleuritic from a pericarditic effusion, especially when no zone of clear lung resonance can be established between the two exudates. As a rule in case of a complication of the two diseases, the occurrence of a pericardial effusion suddenly aggravates the symptoms. Dyspnœa increases to orthopnœa, the heart's action becomes frequent and so insufficient as to lead to sudden distention or continuous undulation of the cervical veins. The pulse is small and the tones of the heart feeble, even in absence of demonstrable dislocation or compression of the heart (Rosenbach).

It may not be denied that notwithstanding all the progress in the study of pericarditis, there are cases where it is impossible to distinguish the friction sound of pericarditis from that of pleuritis, as inflamed surfaces of the pleura may be brought in contact by the action of the heart.

The difficulty of distinguishing pericardial from pleuritic fluid was illustrated by the case reported by Lebric. From a child affected with double pleurisy and pericarditis more than a litre of purulent serum was removed by thoracentesis in the fifth interspace four centimetres outside of the left nipple. The fluid was supposed to have been derived from the sac of the pleura. Autopsy revealed the fact, however, that the fluid had come from the pericardium and not from the pleura, which was adherent to the lung. The case is instructive also as showing the extent to which the pericardial sac may be distended. On the other hand, Desault mistook a circumscribed pleuritic effusion for pericardial dropsy, and actually operated, but a subsequent autopsy revealed the mistake (Roberts).

Equally difficult at times is the separation of pericardial from endocardial sounds. In this distinction it will be remembered that pericardial sounds are not, or are not so distinctly, isochronous with the movements of the heart. Endocardial murmurs suffer no change under pressure or change of posture. Endocardial murmurs are usually propagated and may be heard at a distance in the course of the great vessels. The pericardial friction sound has feeble propagation. Jaccoud said of it, "*il naît et meurt sur place.*" Endocardial murmurs are more frequent at the apex, pericardial murmurs at the base of the heart, though exceptions occur here on both sides. Sounds limited about or heard with the greatest intensity over the surface of the right ventricle are more probably pericardial, as affections of the valves of the right heart are rare. A sound heard over the sternum, or near the left sternal border, belongs probably to the serous pericardium, and if there may be present at the same time an increase of dulness in this region, the diagnosis of pericarditis may be pretty strongly suspected. The sound without the increase of dulness leaves the diagnosis dubious. The galop rhythm, which is probably to be attributed to implication of the myocardium, has also some diagnostic value.

Further, the pulse in pericarditis is usually not at all affected or later on becomes rapid and small, while the pulse of endocarditis is usually at first bounding and arhythmic. Pericarditis is attended with a sense of oppression and pain in the region of the heart, often with dyspnœa, especially upon motion. These symptoms are usually all absent in endocarditis, which is usually distinguished rather by the predominance of palpitation, often in paroxysms.

In differentiation of the dulness of dilatation of the heart from pericardial effusion Schott remarked upon the retraction of the dulness of dilatation in a short time under gymnastics, while the dulness of pericardial effusion remains unaffected.

Endocardial symptoms due to incompetent muscular action—that is, symptoms indicating relative insufficiency—may be overcome by the use of digitalis.

The diagnosis is chiefly determined by percussion. Dulness is appreciated at the complementary spaces, at the base of the heart, as stated, and at the base of the pericardium, under the sternum or to the right of the sternum, and at the region of the fifth intercostal space. The dulness has the shape of a truncated cone. The appreciation of dulness beyond the position of the apex stroke is a sign of great value. Anxiety, pallor, aphonia, cyanosis, vertigo are signs of cerebral anæmia or congestion. Protrusion of the chest, *voussure*, is uncommon in adult life and usually occurs only in the young.

But the character of the effusion, whether serous, purulent, hemorrhagic, etc., may not be determined by the nature of the originating disease. Thus tuberculosis may excite a simple serous as well as a hemorrhagic exudation, rheumatism a purulent as well as a serous effusion, and septic diseases, though they are wont to produce suppuration, sometimes produce only a serous effusion. Barbacci found the Staphylococcus aureus in three cases of diffuse fibrinous pericarditis, the Diplococcus pneumoniæ in one case of hemorrhagic fibrinous pericarditis. Ernst saw the Bacillus pyocyaneus in the fluid of a pericarditis found in the case of a man from whom there had been removed by puncture several times a large quantity of a clear amber-colored alkaline fluid, which coagulated by heat and after sterilization formed a good culture soil for the tubercle bacillus, which was also found in the fluid. Banti found in the exudation of a pericarditis with simultaneous pleuro-pneumonia the Staphylococcus pyogenes aureus and albus but no diplococcus of pneumonia, although the diplococcus was present in the lungs. The presence of tubercle bacilli in the fluid does not necessarily indicate tuberculosis, since they may have penetrated from a neighboring deposit in the lungs.

It is well to remember that a bloody tinge to the fluid does not necessarily indicate a hemorrhagic pericarditis, as the blood may have escaped under puncture from the rich network of vessels in the pericardial tissue.

In all cases of doubt the diagnosis can be established by the use of the aspirator, which determines not only the existence of effusion but also the character of the fluid. The puncture is made by preference at the third or fourth intercostal space, half an inch to an inch from the left border of the sternum. The instrument should in all cases be tested immediately before use and at the same time disinfected by filling the barrel from a vial of absolute alcohol, and the alcohol may be washed out with a two-per-cent. solution of carbolic acid. With these precautions the author has never seen any mishap in the practice of a quarter of a century and as a rule the diagnosis is cleared up literally in a trice.

But, of course, negative findings do not necessarily exclude pericarditis. There may be no effusion; the exudation may be too thick to flow; the needle may become blocked; the pericardium may be unusually thick; the sac may be missed, etc. The deposit of fibrin layers may make the pericardium sometimes an inch thick. This condition is usually found in tuberculous pericarditis. Most instructive in this connection was the case reported by Viry, who found on each side of the pericardium a cul-de-sac which extended to a lower

plane than the central part of the pericardial cavity. An illustrative case of failure to find fluid is mentioned at the close of this paper.

Fürbringer declares that he has made puncture of the pericardium several hundred times and always with satisfactory results. Fürbringer insists upon it that aspiration should be practised after the introduction of the needle, that is, as the needle penetrates the piston should be gradually withdrawn. So soon as the heart is touched the penetration should stop, as while puncture of the heart is innocent penetration of it might be dangerous.

Prognosis.

The prognosis is difficult. In many cases the disease is so light as to pass actually unnoticed; in other cases so grave as to cause death in a short time. Mild cases terminate favorably in the course of a few days so that all the signs may disappear and the patient be restored to perfect health within a week. Effusions of serous character may be entirely absorbed, and in the course of ten days to two weeks disappear to leave no trace.

On the other hand, the course of the disease may be so rapid, as in scorbutic pericarditis, as to take life in twenty-four hours. Andral saw a fatal termination in hemorrhagic pericarditis in thirty-seven hours. Purulent pericarditis may terminate fatally in three or four days. Duchek gives the percentage of recoveries as 48 to 100; Bamberger as 58 to 100; Louis as 66 to 100. Pericarditis is most fatal in the first years of life (Jendrin) and in advanced age. It is, as a rule, more unfavorable in the female sex (Fraentzel), because, according to Raynaud, of the frequency of puerperal infection as a cause. Complications may indefinitely prolong the disease. Thus Wyss recorded a case in which a rib had been eroded, with the establishment of a fistula which remained open for many years.

The prognosis depends chiefly upon the condition of the heart muscle. So long as the action of the heart continues strong, the outlook is not bad. When degenerative changes ensue the prognosis becomes grave with the gravity of myocarditis. Complication with endocarditis (valvular disease) aggravates the prognosis according to the extent and character of the valve lesion. But here, too, it may be said that the immediate outlook depends upon the condition of the heart muscle. When dilatation supervenes the outlook is bad.

The prognosis depends also to some extent upon the character of the cause. It may be cited as a rule, to which it must be remembered there are many exceptions on both sides, that rheumatism furnishes lighter cases, Bright's disease, pyæmia, and scurvy graver cases of pericarditis.

The character of the effusion makes a great difference. Pericarditis without effusion has for the most part a favorable prognosis. These are the cases which often pass unnoticed. The effusion of clear serum indicates a lighter case than the effusion of pus. The presence of blood usually indicates tuberculosis, cancer, scurvy, or some grave affection. Ichor means a communication with the outside air and is of grave prognostic import. The prognosis is determined also to some extent by the duration of the disease. Cases in which the serum accumulates with great rapidity, pericarditis acutissima, have the future determined largely by the time of operative procedure. The prognosis becomes grave in protraced cases on account of implication of the heart muscle. Great effusions give gravity partly by interfering with the action of the heart, but chiefly by mechanical compression of the great vessels at the base.

Prophylaxis.

Pericarditis is best prevented by avoiding the infections, by jugulation of attacks of rheumatism with proper doses of salicylates, by guarding against the exposure which precipitates new attacks, by the timely treatment of tuberculosis, especially with small doses of tuberculin, by the discharge of accumulations of pus as in the treatment of empyema or any other local depot, by rest of the heart as much as possible, by confinement to the house in warm and well-ventilated rooms, if necessary by stay in bed during the course of the various infections.

Treatment.

The rational treatment of pericarditis resolves itself into the consideration of the causative condition. Aside from these circumstances the disease is treated according to its symptoms. There seems to be growing evidence in the belief that pericarditis may be cut short by the application of cold. The cold is best applied by means of ice-bags, which may be suspended just above the chest or applied directly with the intervention of towels or cloths. The ice-bag should under no circumstances be too heavy and should always rest high on the chest, as an ice-bag at the apex or ensiform cartilage may do more harm than good. A good test of the value of this treatment is the subjective sensation of the patient. Lees treated five cases with the application of cold by means of an ice bag, which he declares was liked by all his patients. Where the sensation of cold is agreeable the application does good. Under no circumstances should the ice-bag be continued when the patient complains of the effects. Sometimes in these cases the very opposite treatment is of

value, and cloths wrung out of hot water applied to the upper anterior part of the chest may give greater relief. Inasmuch as most cases of pericarditis depend upon rheumatism, it is advisable to administer the salicylates in dose of grs. v. to x. every two to four hours. Where pain is excessive, resort must be had to opium in some form. Usually Dover's powder in broken doses, grs. iij. to v. every two to four hours, will suffice. When the pain is more extreme morphine may be preferred, if necessary subcutaneously, in dose of gr. $\frac{1}{6}$–$\frac{1}{4}$. The pain which simulates angina may be cut short by the inhalation of a few drops of amyl nitrite, or may be prevented or relieved by the administration of a drop or two three times a day of a one-per-cent. solution of nitroglycerin.

In connection with fever and perturbation of the pulse, no remedy can compare with quinine, which should be given in the dose of grs. iij.–v., three or four times a day. Any weakness in the action of the heart calls for the use of heart tonics and stimulants, especially the tincture of nux vomica, solutions of strychnine, tincture of digitalis, tincture of strophanthus, and sulphate of sparteine, according to the specifications detailed in the treatment of myocarditis. Dropsy is best relieved by the use of calomel in dose of grs. iij. three times a day for several days, or by diuretin in dose of grs. x.–xv., three times a day for a longer period. Diuretin is of especial value as a diuretic and exercises its effect usually in the course of the third day. The dose may reach if necessary grs. xxx. three times per day, preferably in a glass of carbonated water, soda, seltzer, or Vichy water, most conveniently from a siphon bottle.

The great question in treatment is what to do with an effusion. Where the case is mild, as it is usually in the course of rheumatism or a latent tuberculosis, and the condition of the patient is good, the effusion may be left to itself, as it will probably disappear by absorption in the course of one or two weeks. But when the effusion accumulates rapidly, or when it remains too long, it should be withdrawn by aspiration.

Paracentesis of the pericardium was originally proposed by Riolan (1649), but was first successfully performed by Romero, of Barcelona (1819). The great respect for the heart entertained by the authors of antiquity is responsible for the fear of performing the operation, as it was thought that any accidental injury of the heart substance itself would be necessarily fatal. In this way may be understood the opposition of the surgeons even in the middle decades of the present century. Thus when Billroth, as late as 1870, characterized the operation as a surgical frivolity and declared that the operation seemed like a prostitution of surgical skill, we are reminded

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of the opposition of anatomists, such as Hyrtl, to opening the abdominal cavity. The more accurate knowledge of the nature of the disease, the greater ease in making a diagnosis, the use of aseptic instruments, and the impunity with which the operation has since been done, have made paracentesis a safe, justifiable, and at times an imperative procedure.

Riolan first suggested perforation of the sternum at the xiphoid cartilage with a view probably of getting as far from the heart as possible. This was in the days of the trocar and cannula. The more modern operation of aspiration, which secures the withrawal of the fluid through a small hollow needle, has enormously simplified the process and minimized the dangers. No real damage is done even if the heart be touched with the point of a small needle. Hulke once tapped the right ventricle and withdrew one drachm of blood without the least bad effect. In fact, the abstraction of blood seemed to relieve the distended heart more directly than a venesection.

Steiner found in his experiments with electro-puncture that needles could be introduced into either ventricle with perfect impunity provided they were at once withdrawn.

Penetration is now made by most operators at a point half an inch to an inch from the left border of the sternum, anywhere from the third to the fifth intercostal space, as determined by dulness and sometimes by subcutaneous œdema.

Rosenbach recommends as the point of election for puncture the fourth or fifth intercostal space about one centimetre outside of the mammary line. There is here, he maintains, no danger of wounding the heart with a fine cannula. Dieulafoy aspirates at the fourth or fifth intercostal spaces six centimetres from the edge of the *left* border of the sternum. Wilson twice practised paracentesis of the pericardium through the fifth right intercostal space, penetrating one inch from the *right* border of the sternum with an ordinary aspirating needle and evacuating 15 to 20 ounces of serous fluid, with immediate relief of the severe symptoms. West (1883) reported from the literature 79 cases with 36 recoveries, in which the point of election for puncture was in the fifth intercostal space, one inch from the *left* border of the sternum. Siewers reports 4 recoveries in 9 cases, and selects as the point of puncture the fourth or fifth intercostal space, two centimetres to the *left* of the sternum.

The operation is now done frequently, in fact, more frequently than is believed or reported. Thus when Schrötter, in his recent communication, speaks of the report of 100 cases, he has certainly collected from a comparatively narrow field of literature, though he quotes from Weismayr, who took the trouble to look up all the

recorded cases to the number of 99, to which he adds the latest report by Mader, which made an even 100.

Different authors give different statistics as to the result of these operations, but statistics in this connection are of not much value because so much depends upon the character of the effusion, and more especially upon the condition of the heart muscle. Thus in two unrecorded cases in the practice of the writer of this article, paracentesis was followed by immediate relief, in one case to such an extent that a boy, who had been confined to his bed unable to raise himself on account of dyspnœa for two weeks, was able to leave the house and resume his occupation in the course of eight or ten days. Subsequent accumulation necessitated repeated puncture, and the boy finally succumbed to a sudden heart failure. In nearly all cases the immediate relief is pronounced. Rendu reports permanent improvement in thirty-two per cent., and in 8 out of 10 cases affected with uncomplicated pericarditis there was absolute recovery. Of the cases collected by Weismayr, including Mader's, 47 recovered, 53 died.

Grainger Stewart reported of 97 cases 38 recoveries. This percentage of recovery would be greatly increased if the cases of scorbutic, hemorrhagic, and purulent pericarditis had been classified apart, as these cases require not simple puncture but incision and drainage. The separation of these cases leaves 65, of which 30 recovered absolutely, 13 were improved, and 22 died. The deaths could not in any way be attributed to the operation, though in two cases injury to the heart had been followed by subsequent hemorrhage. In all the other cases death was due to the original disease or to complications—tuberculosis, heart lesions, pleurisy, etc.

Reaccumulations necessitate a repetition of the operation. Moor operated six times in the case of a boy aged thirteen. Bouchut, in the case of a girl aged twelve, operated eight times. Schrötter reported a case of hemorrhagic pericarditis with serous effusion in the right pleura, in which he performed aspiration of the pericardium thirteen times and paracentesis of the pleura seven times. This patient lived over a year. Churton reported thirteen punctures for relief of hemorrhagic effusion in a man aged forty-six; a final relapse was followed by death. In the case reported by Schuh, the patient, who could neither sit down nor lie down on account of extreme dyspnœa, felt perfectly well after the operation and was discharged cured in twenty-one days. This patient returned, however, six months later and succumbed to carcinoma of the mediastinum. The autopsy showed that the operation had been a perfect success, in that it had secured adhesion of the layers of the pericardium and obliteration of

the sac. Audeoud also reported a case of paracentesis in hemorrhagic pericarditis followed by complete recovery, though death occurred three months later on account of perforations of the intestine from tuberculosis of the abdominal viscera. The autopsy showed that the heart had regained nearly a normal appearance. In all cases the fluid should be withdrawn slowly, but not wholly; that is, there should be no attempt to secure the discharge of the last drops.

When the effusion is purulent, and still more when it is ichorous, the pericardium must be drained and the sac treated precisely as an empyema. Siewers considers drainage necessary, but advises that irrigation should not be done. Repeated or rapid reaccumulation may be prevented by the injection of irritants, as by solutions of iodine. Rosenstein, West, Partzewski, Bronner, Davidson, Koerte made extensive resections. The operation was always attended by relief, but the patient did not always recover because of irremediable lesions or complications. In this operation the incision is made close to the left border of the sternum, and the tissues are divided layer by layer until the pericardium is reached. The pericardium is then dissected free, the external layer lifted with a pair of forceps and divided. Eiselberg, who operated successfully on a case of traumatic purulent pericarditis by incision after exploratory puncture and irrigation with warm salicylated water, insists upon the importance of suturing the pericardium to the lips of the incision to prevent infection of the pleura. In a case which he reported complete recovery took place in four weeks. Fraentzel condemns this operation as unnecessary, as he declares that no one dies of pure pericardial exudation. He bases his belief upon the condition of a case which showed threatening symptoms for a long time, and subsequently recovered without an operation. Nevertheless, the results which have been obtained in obstinate cases of empyema would seem to point in similar affections of the pericardium to the necessity of more radical operation than a mere aspiration.

Negative findings, as stated in the practice of aspiration, do not necessarily exclude the presence of fluid in the pericardial sac. Potain reported a case in which no fluid escaped, though the instrument penetrated until the beat of the heart could be felt. When it was withdrawn it was found that the needle was blocked with a piece of false membrane. The patient died six days later, and at the post-mortem it was seen that the pericardium contained a litre of sero-purulent fluid the discharge of which might have turned the scale in his favor. For it must be admitted that, in the vast majority of cases, aspiration properly practised, and if necessary repeated or followed by the injection of irritant substances to secure obliteration

of the sac, will control the situation so far as pericarditis is concerned. The condition of the heart muscle or the presence of any causative or associate disease will require consideration of itself.

Concretio Pericardii.

Concretio pericardii, cardiac symphysis, obsolescence of the pericardium, was regarded by the older writers as a condition of great gravity. Columbus, Bartholin, and Tulpius considered it a congenital defect, an error which was corrected later by Lancisi and Haller. Morgagni (1762) and Lieutaud (1767) collected all the cases published up to their day. By this time it was known that the condition represented only a particular termination of a common pericarditis, in which apposed inflamed surfaces become adherent, so that the concretio pericardii is to be regarded only an adhesive pericarditis, and the consideration of it in a separate section is really a deference to ancient views.

Termination by adhesion is not rare, but the condition is usually discovered only on autopsy. Thus Leudet found it 61 times in 1,003 cases. The statistics from the Berlin Charité show 156 cases of adhesion of all kinds in a total of 324 cases of pericarditis. Adhesion may occur at any age, but has been met more frequently in advanced life. Bednar found it in an infant of three months and Billard and Hunter in new-born children as evidence of pericarditis during fœtal life. Geist found it 26 times in 514 autopsies of old people, aged sixty to ninety-three. As may be readily understood the adhesion is more frequently partial than complete, and in fact general adhesion is more frequently apparent than real, as in many places the membranes are merely apposed and not adherent. Leudet reports partial adhesion 5 times in 100 cases, and total adhesion 2.5 times in 100 cases. Partial synechiæ are found most frequently in the neighborhood of the great vessels.

Sometimes the adhesion is immediate, that is, apposed surfaces lie in direct contact. Sometimes the surfaces are connected by shaggy, hairy, or villous processes or by bands of greater or less strength. Sometimes the process of adhesion is affected by the interposition of chalk plates, which were formerly considered as ossifications or calcifications of the pericardium, but which are now more properly interpreted as cretifications or calcifications of the exudation.

Thus Rivet found on autopsy in a woman aged seventy-four, who had suffered in life general dropsy with regular but weak action of the heart and had shown muffled sounds but no murmurs,

the hypertrophied heart enclosed in an outer casing of chalk of the thickness of a centimetre, with free spaces only at certain spots. The reduced action of the heart which goes with the further course of pericarditis is necessary to this deposit (Schrötter). See also other cases in the section on Pericarditis.

Adhesion may occur under any kind of inflammation and before or after effusion. The process of calcification, according to Richards, is always consecutive to suppuration with subsequent absorption and cretification. So adhesion may occur at any period in the course of pericarditis. Sometimes it develops rapidly. Cerf found evidence of adhesion in nine days, and Bouillaud, in an autopsy which was made in twenty-four days after the inception of the disease, saw adhesion "which could with difficulty be torn asunder."

Symptoms.

The fact that most cases are discovered only on autopsy proves that the condition is often latent. In fact, it is known that entire obliteration of the pericardial sac may remain without clinical evidence, and the grave symptoms ascribed to the condition by the older writers are referred in our day wholly to complications, chiefly on the part of the myocardium. Laennec declared that he had opened a large number of bodies of individuals who had never made complaint of any trouble with respiration or circulation, where there was found an intimate and total adhesion of the lungs or of the heart, "but as regards this latter organ in particular I am constrained to believe, after having encountered a number of cases of this kind, that adhesion of the heart to the pericardium in no way interferes with the exercise of its functions. It would seem to me only that the contraction of the auricles may become somewhat obscure when they are adherent to the fibrous layer of the pericardium." It was the recognition of this fact that led Laennec to put the proper estimate upon pericarditis in general. Thus, "these facts and several others appear to me to prove that in certain cases acute pericarditis is a local affection of little gravity, whose influence upon the system in general and even upon the circulation is almost nil."

It might be inferred that adhesion of the heart to the pericardium would interfere with the free movements of the heart, but no such evidence is found in fact; and the symptoms which have been, and are yet to some extent regarded as characteristic, are to be referred rather to traction upon the great vessels or interference with the circulation of blood in them.

Among these symptoms may be considered first the peculiar

change which takes place in the pulse during inspiration. It was observed first by Hoppe (1855), next by Griesinger, that in certain cases the pulse becomes reduced in volume or disappears entirely during the act of inspiration. As the act of inspiration, by expanding the thorax and relieving pressure on the great vessels, should rather favor than hinder the efflux of blood from the heart, the reduction in volume at this time was considered anomalous, and the condition was distinguished by Kussmaul as the *pulsus paradoxus*. This paradoxic pulse was observed in certain cases of concretio pericardii, and was for a time considered as characteristic of the condition. It was soon seen, however, that the paradoxic pulse occurs only in cases in which there is some interference with the efflux of blood from the heart, and the condition shows itself in pericarditis only when the adhesions are so situated as to make traction upon the great vessels at the base. This traction or retraction can be thus efficacious when the layers of the heart are not only united with each other, but when the outside layer is also united to the thoracic wall, so that the paradoxic pulse speaks rather in favor of a mediastinal pericarditis than of a mere concretion of the pericardial layers. Thus Wiedemann described a case of paradoxic pulse in a severe pericarditis in which the aorta was so firmly bound down by exudation to the mediastinum and sternum that with every inspiration it was drawn forward, bending at an angle, and had its lumen greatly reduced. Kussmaul also connected the paradoxic pulse with cicatricial mediastinal pericarditis, and Boehr and Herrlich found it in a case of exudative pericarditis with mediastinitis. Subsequent observations have shown, however, that this pulse may occur with any condition which interferes with the escape of blood from the heart, entirely independent of pericarditis of any kind. Thus Bauer found the paradoxic pulse in a case of large pleuritic exudation on the left side.

The most striking and obtrusive abnormality considered in this connection is the sinking in of the chest-wall in the region of the apex, especially of the intercostal space, with each systole of the heart. This condition was attributed directly to adhesion of the pericardium. The fact was established even by Harvey, that when the heart is free and the pericardium in a normal state, the systole is attended by protrusion of the intercostal space, as in its contraction the apex is tilted forward to strike against the wall of the chest. It was thought that when the pericardium became adherent this normal motion would be interfered with to such extent as to produce retraction rather than protrusion of the wall of the chest, and for a time this sign was considered as pathognomonic of pericardial adhe-

sion. It was soon seen here too, however, that retraction occurs only when, together with adhesion of the two layers of the pericardium to each other, the parietal layer is itself adherent to the wall of the chest, so that retraction of the chest is indicative again rather of mediastino-pericarditis than of a mere concretio pericardii. Moreover, Bahr saw systolic retraction in a case entirely free of adhesions, and Friedreich made the same observation in a case of stenosis of the aortic orifice with consecutive hypertrophy of the left ventricle. Retraction of the intercostal spaces is natural at the base of the heart and may be always observed, when the movements of the heart can be at all distinctly seen, at the right and left of the sternum. Retraction must necessarily occur in all regions in which the heart is not covered by the lung. When the heart contracts it shrinks in volume in every direction and atmospheric pressure forces in the wall of the chest, unless the space formerly occupied by the heart is substituted by the lung. The base of the heart lies in immediate apposition to the chest-wall, and these retractions are sufficiently explained by atmospheric pressure.

It was further believed that adhesion of the pericardium would so interfere with the action of the heart as to lead to consecutive hypertrophy. Hope held that long adhesion produced hypertrophy on account of the strain thrown upon the heart, but Gairdner showed by statistics that this view is incorrect and that hypertrophy when present is due to other cause. The fact is that hypertrophy is only rarely observed in concretio pericardii. Adhesion of the pericardium does not throw extra work upon the heart. Hypertrophy occurs only in the face of obstacle, as in the case of valve lesion, increase of blood pressure, etc. Changes which occur in the heart muscle in connection with concretio pericardii are to be referred to associate disease or to disease causative of both conditions, endocarditis, Bright's disease, etc.

The condition of the veins in the neck has been considered as a sign of some value. It has been noticed that the veins are distended during the systole, and are so suddenly emptied during diastole as to collapse and disappear. Sometimes the collapse is so complete as to lead to retraction of the superjacent tissue. But this sign indicates adhesion of the pericardium only when the pericardium is adherent to the wall of the chest. The expansion of the chest draws upon the blood in the cervical veins so powerfully as to suddenly empty them.

The fact is, therefore, that a mere cohesion of the parietal and visceral layers of the pericardium may be unattended with any evidence of positive character, and the condition, as stated, is oftenest discovered only upon autopsy.

But adhesion of the two layers of the pericardium is usually attended with evidence of disease of other parts of the heart, as of the heart muscle, of the endocardium, etc. These conditions make themselves manifest by distinctive signs. The symptoms present are therefore due, not to the adhesion of the pericardium, but to the associate disease, and the reason why severe symptoms are shown in some cases and not in others is, as was shown long ago by Kreysig, because in some cases the heart muscle is gravely, and in others lightly or not at all affected.

Mediastino-Pericarditis.

When the outside layer of the pericardium is united to the wall of the chest or to the mediastinal tissue, the symptoms become more distinct. This condition is seen most frequently in cases of more extensive inflammation and is probably more distinctly connected with tuberculosis than any other single affection. As a rule, the pericardium itself shows marked change. Sometimes the two layers are adherent. The outer layer is very much thickened and masses of connective tissue unite it with the sternum. Hence this form of inflammation is often known as "indurative." Sometimes the adhesion is more mediate, in the formation of bands, and these bands may extend along the tract of the aorta and great vessels, subjecting them to compression or torsion, especially during acts of inspiration. Or the adhesions may extend downward and laterally to connect the pericardium with the diaphragm and pleura. A striking illustration of an extensive affection of this kind was reported by Claessen, who described a case of tuberculous cicatricial mediastino-pericarditis with adhesion to the diaphragm, the origin of which was not clear, but could probably be referred to a caseous bronchial gland. The affection extended to the pericardium and led to complete adhesion with the heart, then excited myocarditis and developed a tumor which protruded into the right auricle and almost entirely blocked the orifice of the superior vena cava which resulted in heart failure and extreme stasis in the domain of this vessel.

Symptoms.

Adhesion of the pericardium with the mediastinum or with the wall of the chest interferes with the expansion of the lungs so that the area of absolute heart dulness is increased. Usually as the lung expands in inspiration, it insinuates itself in front of the heart. Adhesion of the heart would necessarily interfere with this excursion of the lungs. As the act of inspiration expands the chest-wall in every

direction, the heart must fill the space which would otherwise have been occupied by the lungs, so that in cases of extensive adhesion the right heart may suffer such distention as to lead to relative insufficiency of the tricuspid valves. The most obtrusive evidence which presents itself is, however, the systolic retraction of the chest-wall.

Mediastino-pericarditis proper implies adhesion at the base and not at the apex, so that this retraction, as stated, is a sign simply of concretion of the heart with the wall of the thorax.

The *pulsus paradoxus*, that is, the interruption of the pulse by inspiration, has been noticed most frequently in connection with mediastino-pericarditis, the explanation being that the aorta suffers compression or traction from the bands of connective tissue which bind down the great vessels. It may be understood, however, that any fixation of the body of the heart would have the same effect. Thus, for instance, adhesion of the pericardium with the diaphragm would necessarily make traction upon the aorta.

The *pulsus paradoxus* has been noticed, however, in so many conditions as to possess but comparatively little value in the diagnosis of mediastino-pericarditis. It has been noticed, as already stated, in the presence of complete concretio pericardii, and sometimes in the presence of great effusions. Schrötter reported a typical case in a young girl affected with cirrhosis of the liver, with probable concretio pericardii. Kussmaul observed swelling of the jugular veins with every act of inspiration, and ascribed it to traction upon the vena cava or innominate vein.

Most of the cases of mediastino-pericarditis show some of the symptoms of pericarditis; dyspnœa, palpitation, arhythmia, weakening of the apex stroke; sometimes with evidence later of effusion which has become absorbed or sacculated. No single symptom is, therefore, indicative of mediastino-pericarditis, but the association of a number of symptoms may justify a diagnosis. Thus the coincidence of the paradoxic pulse with inspiratory swelling of the cervical veins has never been observed in a simple pericarditis.

The general symptoms of adhesion of the pericardium may be practically divided into two classes, the mediastinal and the myocardiac. The mediastinal symptoms are, as stated, retraction of the chest-wall, sometimes the paradoxic pulse, swelling of the cervical veins during inspiration, fixation of the border of the lungs. The myocardiac symptoms are weakening and irregular action of the heart and pulse, evidence of degeneration and dilatation in increase in the area of dulness, insufficient blood supply, especially to the brain and kidneys, and dilatation which may be so extreme as to lead to relative insufficiency of the tricuspid valve.

Pins once heard a musical murmur which he attributed to tension of a fibrous cord passing between the aorta and the great bronchus. The fact that this murmur was strongest on inspiration and was associated with a paradoxic pulse, made the explanation plausible.

Diagnosis.

Tumors of the mediastinum which might most closely simulate an extensive indurative inflammation may be differentiated by the increase in the area of dulness, dislocation of the heart, and enlargement of the lymph glands. Tumors of the mediastinum often show also ectasia of the veins of the surface and do not show the paradoxic pulse. As carcinoma is the most frequent tumor, this condition may manifest itself in the pain, cachexia, and other signs characteristic of malignant disease. Aneurysm of the aorta would show a characteristic thrill and bruit, and would in the course of time erode the wall of the chest.

Treatment.

The treatment must address itself to the cause of the condition, to the relief of tuberculosis, where this condition can be demonstrated to exist, especially by the use of small and gradually increasing doses of tuberculin.

Cantlie recommended body movements in the beginning of convalescence of pericarditis to prevent adhesion, but as some other critic has already remarked, this recommendation would certainly seem to have more theoretical than practical value.

Hydropericardium (Hydrops Pericardii).

Hydropericardium implies an abnormal increase in the pericardial fluid. The amount of fluid which is considered normal is variously stated. Perhaps it might be true to say that this amount varies within certain ill-defined limits. In all cases the fluid is present in sufficient quantity to moisten the membrane, and in most cases an appreciable amount can be collected. Hammerston, who made his studies in the bodies of individuals who had been executed, found that he could get a quantity sufficient for chemical tests. The fluid may accumulate in extreme cases, to lead to distention of the pericardial sac. But these great accumulations are very rare.

The pericardial, like other serous effusions, is a clear, amber-

colored, alkaline fluid, containing salts, urea, sometimes traces of sugar, and, according to Hammerston, more fibrinous matter than other transudations. The clear fluid is sometimes rendered slightly turbid by cells of epithelium, floccules of fibrin, and occasional red blood corpuscles.

Hydrops pericardii is an expression of retarded or interrupted circulation. The condition is usually encountered, therefore, in connection with general dropsy and is most frequently seen in the last stages of Bright's disease and valvular lesions of the heart. Hydropericardium is observed also in the hydræmic states, which occur in marasmus from any cause, especially from tuberculosis, carcinoma, Addison's disease, diabetes, etc., though in these cases the fluid never accumulates in the quantities encountered when there is an interruption of the circulation. Sometimes the interruption is from local cause, as from interference of the circulation of blood in the coronary vessels themselves.

Hydropericardium occurs with the other transudations in beriberi and as frequently as ascites, that is, in thirty per cent. of cases.

Hydrops pericardii is not infrequent, being found, according to the statistics of Günzberg and Duchek, from seven to fifteen times in one hundred autopsies.

Symptoms.

Hydropericardium develops, as a rule, insidiously, and the presence of the condition may be prediacted in cases where the fluid has gradually mounted up the lower extremities to invade the peritoneum and the pleura. Attention is directed to the probability of effusion by the condition of the heart, which is found in a state of degeneration with dilatation of its cavities. The fluid accumulates as in cases of exudation from pericarditis, that is, first in the complementary sinuses. In extreme cases, as stated, the pericardium may be distended though never to such a degree as in pericarditic exudations. Thus one litre would represent the maximum quantity of hydrops pericardii.

Inspection reveals no change as a rule. Anything like a protrusion or a vaulting of the chest is absent. The apex stroke usually falls in the normal site. The strength of the stroke will vary according to the condition of the heart itself. The feeling as if the heart struck the wall of the thorax at different times in different places, mentioned by Testa, subsequent observers have never been able to discover. Percussion discloses the outlines of the pericardium. Sometimes, as in pericardial effusion, dulness extends beyond the apex stroke. Great accumulations depress the diaphragm, compress

the lungs, and show interference with the action of the heart in dilatation of the cervical veins, cyanosis, and dropsy. Auscultation reveals more or less muffling of the heart sounds, dependent, however, rather upon weakness of the heart muscle than upon accumulation of fluid. The impact of the heart is changed with the posture of the patient. In the presence of great effusions it becomes muffled and indistinct. Signs of heart failure are revealed in insufficient blood supply of the brain and kidneys. The patient suffers attacks of vertigo and faintness; the urine is scanty, high-colored, and shows heavy deposit.

Diagnosis.

Hydrops pericardii, like pericardial effusion, must be differentiated first from an enlarged heart. When the enlargement is due to hypertrophy the force of the heart is the feature of importance. The separation of a dilated heart is sometimes more difficult, especially as the conditions so often coincide. The existence of endocardial bruits, especially of mitral lesions, may determine the diagnosis. Dulness beyond the apex stroke speaks for hydropericardium, as does also change in the force of the impact, with change in the posture of the patient.

Aneurysm of the aorta may be differentiated, in the absence of any visible pulsating tumor, by the existence of the bruit and thrill characteristic of aneurysm, as also by the dislocation of the apex in aneurysm on account of elongation of the aorta downwards and to the left. Aneurysm is most common in connection with the history of syphilis and with a life of strain and hard work. Tumor of the mediastinum is most commonly cancerous, occurs at a later period of life, and is attended with pain. These tumors usually dislocate the heart. Encapsulated pleuritic effusions may usually be differentiated by the history of previous pleurisy, which in turn is dependent most frequently upon tuberculosis and pneumonia. Hydropericardium belongs more especially to Bright's disease and to lesions of the valves of the heart.

The real difficulty of diagnosis occurs in connection with pericarditic effusion. Pericarditis as an active disease of itself is usually attended with pain, sometimes with tenderness, and occurs as a link in the chain of an infectious process, rheumatism, pneumonia, tuberculosis, etc. In cases of insidious origin and in the absence of definite history, which might establish a genetic relationship, the differentiation becomes more difficult. In all cases of doubt as between effusion of any kind and enlargement of the heart itself the question may be settled by aspiration. But aspiration would with-

draw fluid from both a pericarditic exudation and a hydropericardial effusion. Nevertheless the fluid of the exudation and that of the transudation may show distinctive peculiarities. The transudation of hydropericardium usually has a light specific gravity, 1.005–1.012, shows less chlorides and decidedly less albumin. The exudation of pericarditis may contain as much as seven per cent. of albumin. Where the albumin amounts to three per cent., there is or has been nearly always inflammation, that is, pericarditis. The exudation of pericarditis is, when serous, perfectly clear, but it may become more turbid as the fluid is withrawn. It contains more albumin, more fibrin, more white blood corpuscles, and more floccules of epithelium.

In spite of all these distinctions the diagnosis may be difficult. Very welcome, therefore, was the contribution of Rosenbach and Pohl of a sure means of separating hydrothorax from pleuritic effusions, which may be utilized in the distinction of the analogous affections of the heart. These observers found that small quantities of iodine and iodide solutions administered by the mouth passed rapidly into the transudations in large quantity, while they could be found only in traces in the inflammatory exudations. In fact, in the exudations the quantity is so little as to necessitate desiccation of the fluid to detect it at all. The test may be made with the small amount of fluid withdrawn by the hypodermatic syringe in the recognition of the presence of fluid. If a few drops of fuming nitric acid be added, and the fluid be agitated with chloroform, the presence of the red color of iodine, which sinks to the bottom with the chloroform, indicates a transudation. A mere trace of color would indicate a complication of exudation with transudation.

Prognosis and Treatment.

The prognosis of hydropericardium depends wholly upon the originating malady. It is usually bad. The pericardium is invaded, as a rule, only in the later stages of the original malady, and hydrops pericardii is usually found in connection with degeneration of the heart muscle itself. Nevertheless, even large effusions may be absorbed, the heart muscle be restored, and individuals may recover perfect health. Such a favorable result is most frequently accomplished in cases of Bright's disease, where the heart muscle is comparatively sound, and is usually reached through stimulation of the heart with diuresis. Thus the exhibition of calomel in dose of grs. iij. three times a day for several days may lead to the absorption of large effusions. The administration of diuretin in dose of grs. xv. in a glass of soda water three times a day may have the same effect.

Large effusions may be made to disappear rapidly with the other signs of dropsy under the use of the infusion of digitalis in the dose of ʒ i. to ʒ iv. with the acetate of potash, grs. v.–xv., three or four times a day. The treatment of hydropericardium really resolves itself into the treatment of the associate weakness of the heart itself.

Hæmopericardium.

The term hæmopericardium is limited to effusions of blood into the pericardial sac. The condition excludes hemorrhagic pericarditis, which is usually found in tuberculosis, cancer, more especially in scurvy or other cachexia, sometimes, more rarely, in the alcoholic pericarditis of old people. Hemorrhage into the pericardium occurs most frequently, aside from penetrating wounds, including fractures of ribs, of the sternum, etc., from rupture of the heart, more rarely from rupture of an aneurysm of the aorta or of the coronary artery. Eichhorst reported a fatal hemorrhage from a tuberculous ulcer. The quantity of blood varies. Simple extravasations may stud the surface as ecchymoses. Such exudations are found in the extreme venous stasis of strangulation, sometimes as the result of grave infections, more especially in phosphorus-poisoning. Sometimes only the coloring matter of the blood is found staining the natural effusion. On the other hand, blood may accumulate in such quantity as to distend the pericardial sac. The almost incredible amount of eight pounds reported by Corvisart was found in an exudation stained in this way. The blood is sometimes found free and fluid, sometimes coagulated *en masse* or in layers. In a case in the practice of the writer there could be stripped from the pericardium several successive layers of blood which had escaped from a rupture of the heart substance in small quantities at a time, the patient having lived for several days after the rupture.

Hæmopericardium is even more grave than hemorrhagic pericarditis, as independent of the originating malady the effused blood interferes with the action of the heart.

Symptoms.

The symptoms vary in every degree of intensity. When the hemorrhage proceeds from rupture of the heart, the accident is announced by the sudden occurrence of agonizing pain, with pallor, prostration, and collapse; sometimes there are convulsions. The patient succumbs to shock, or he may survive to succumb more gradually under the signs of hemorrhage with anæmia of the brain and syncope, or

with dyspnœa and cyanosis from direct interference with the action of the heart. Not infrequently the patient is suddenly transfixed with pain, the face becomes livid, and death is almost immediate. Rupture of an aneurysm of the aorta would have a history of this kind, but rupture of a smaller aneurysm of the coronary artery or of one of its branches would discharge smaller quantities of blood and be unattended with the same sudden danger.

Diagnosis.

The diagnosis is usually obscure and is often established only upon autopsy. The recognition of the accident depends upon (1) the nature of the originating malady, trauma, myocarditis, aneurysm, etc.; (2) the signs of shock or of hemorrhage; (3) the interference with the action of the heart; (4) the increase in dulness with disappearance of the apex stroke and muffling of the heart sounds; (5) the withdrawal of fluid by aspiration. This suggestion is largely theoretical, as most cases of blood withdrawn from the pericardium belong to hemorrhagic pericarditis. Negative findings would not exclude hæmopericardium, as the blood may be coagulated, so that this test is of value only in quite recent cases.

Hæmopericardium is differentiated from hemorrhagic pericarditis by the genetic relations of the two affections. Hæmopericardium belongs especially to trauma, rupture of the heart muscle, or rupture of an aneurysm. Hemorrhagic pericarditis is the form of pericarditis which is developed most frequently in tuberculosis, cancer, scurvy, or diseases attended with disorganization of the blood.

Prognosis.

The prognosis is always grave, but depends upon the cause of the condition. Death may occur immediately or in the course of a few hours or days. Nevertheless recovery is possible after the escape of but small quantities of blood, in conditions which are in themselves not incurable.

Treatment.

The treatment is directed wholly to the cause. It resolves itself, from a medical standpoint, into treatment of rupture of the heart, rupture of an aneurysm, etc., that is, to the treatment of internal hemorrhage. The transfusion of one-half pint to a pint of the physiological solution of salt (0.6 per cent.) would be the remedy of most value.

Pyopericardium.

As in the case of the effusions of blood, pyopericardium must be distinguished from purulent or suppurative pericarditis, and the term should be limited strictly to cases which occur independent of infectious disease, as by discharge of pus from an abscess of the heart muscle or by irruption of an abscess from some contiguous viscus. The fact is, there is so little ground for separation of the two conditions that they are usually considered together. Purulent pericarditis proper occurs withe special frequency in pyæmic processes, in croupous pneumonia in children, and in drinkers. Pyopericardium occurs at any period of life independent of or in connection with suppurative inflammation in some contiguous organ. In other respects the diagnosis, which may be determined by aspiration, does not differ from that detailed under purulent pericarditis.

The *prognosis* would depend altogether upon the cause of the condition. Aside from this fact, the outlook is in a general way not unfavorable, as a pyopericardium is *sensu strictu* a localized abscess.

The *treatment* demands imperatively incision and drainage with sustentation of the heart.

The following are interesting recent contributions:

Davidson gives the details of a case in which an incision was made into the pericardium and eight ounces of pus evacuated and a drainage tube three and one-half inches long was inserted. Relief was afforded, but the patient died seven days afterward. Davidson records another case in which pyopericardium was complicated with empyema in a child six years old, and an incision into the pericardium with the use of the drainage-tube was entirely successful.

Scott evacuated pus from the pericardium in a child aged six by incision and drainage, with subsequent irrigation of the sac with a one and one-half per cent. solution of carbolic acid. The boy made an uninterrupted recovery.

A remarkable case of empyema of the pericardium was published by Dickinson. In a boy aged ten years there was developed, in connection with a large abscess in the sacrum, which opened spontaneously, a considerable serous effusion in the left pleura, purely of pyæmic origin, and an empyema of the pericardium. There was pronounced œdema of the thorax, ectasia of the veins of the lower part of the chest, and swelling of the liver. The pleuritic exudation was removed by aspiration twelve times without ever assuming a purulent character. The fluid in the pericardium was removed three times by puncture and aspiration with a discharge of 45, 350, and 580 grams

respectively. The operation was done on account of threatening symptoms. But as the pus continued to form, an incision was finally made with the knife and a rubber drainage-tube inserted. The case terminated in perfect recovery in two and one-half months. The puncture was made in the fifth intercostal space, to the right of the sternum and close to its edge.

A more extensive incision was made by West, in the case of a boy aged sixteen, from whose pericardium pus to the amount of fourteen ounces was first withdrawn by the trocar. In a few days the same amount was again taken away, but the fluid still reforming an incision was made into the sac, through the fifth interspace, and a drainage-tube inserted; two quarts of purulent fluid were then removed. The patient gradually recovered and remained quite well. Gussenbauer once resected five ribs, drained the cavity of the pericardium, and washed it out with a disinfecting solution (thymol), rescuing the patient absolutely.

Pneumopericardium.

Pneumopericardium, pericardiac pneumatosis, is an accumulation of air or gas in the cavity of the pericardium. But air never exists alone. It would be impossible to conceive of the presence of air or of gas in the pericardial cavity without fluid of some kind. The condition is then named, according to the character of the contained fluid, hydropneumopericardium, pyopneumopericardium, hæmopneumopericardium, etc. The fact is the effusions are nearly always purulent or ichorous, and purely serous effusion, such as is sometimes seen in pneumothorax, has been reported only once, by Müller, in pneumopericardium.

Pneumopericardium was seen by the older anatomists and clinicians, especially by Sénac and Morgagni, and was distinctly recognized by Laennec and Bouillaud. Bricheteau (1844) described the sound of concussion. The condition has been since observed, and the reported cases have been collected especially in the French encyclopædias. But cases of pneumopericardium are rare. Thus Schrötter never saw a case in the vast material of the Vienna Hospital in thirty-five years, and Skoda, in all his observations in the same place, had the same experience. Liebermeister declares that he saw but one case of pneumopericardium and then when an assistant in the clinic of Niemeyer; this was a case of perforation by carcinoma of the œsophagus subsequently described by Tütel. The writer of this paper has never seen a case of pneumopericardium here or anywhere in this country or in Europe.

The presence of air or gas in the pericardial cavity implies penetration from without, including in this statement communication with some viscus which contains air, the lungs, the stomach, etc. The older writers believed in the spontaneous generation of gas in the pericardial sac. The possibility of such a condition may not be denied. It is certainly true that gases are developed in the products of decomposition. It is therefore possible to conceive of a pneumatosis in the course of a purulent pericarditis, but this possibility implies that secondary change of pus into ichor, which results only after admixture with the bacteria of decomposition. The fact that gas has been found in the cavity while the most careful examination failed to disclose any opening, would seem to prove a local development. Cases of this kind were reported by Bricheteau, Stokes, Duchek, Friedreich, and others. These cases are better explained, however, by assuming a closure of a pre-existent opening, so that the idea of a spontaneous development of gas in the pericardial sac has but few advocates in modern times. Heufner analyzed the gases from a pyæmic abscess in the pericardium with the following result:

Carbonic acid gas and sulphuretted hydrogen, 1.05 per cent.
Oxygen..... 14.50 "
Nitrogen..... 84.45 "

He concluded from this chemical composition that the gas must come from the atmosphere. Nevertheless the existence of gas in the interior organs, entirely shut off from outside communication, as for instance in a thyroid gland, leaves the question still unsettled, and as Dressler in his analysis found more carbonic acid gas and less oxygen, and believed, therefore, that gas was spontaneously developed, there is still room for doubt. The following, reported by Friedreich, is an illustration of such a case. A strong, previously healthy servant girl, thirty-three years of age, was brought to the lying-in institute in labor two days, and was delivered of twins. The second child had to be turned. The woman was subsequently attacked with puerperal fever, apparently of pyæmic origin, and died. Pleurisy had developed on the left side with rapid exudation. The heart was dislocated to the right. There was intense dyspnœa. Pneumopericardium set in in the course of several weeks and announced itself with tympanites in the region of the heart. Clear, metallic heart sounds like the notes of a zither could be heard some distance from the patient. The diagnosis of pyopneumothorax and pyopneumopericardium was verified on autopsy. Puncture of the pericardium discharged a quantity of fetid gas as well as a considerable quantity of ichorous matter. As the most accurate investigation failed to find any perforation of the pericardium, Friedreich consid-

ered it a case of spontaneous development. Among the most recent writers Leube declares outright that there are certain authentic cases of accumulation of gas in the pericardium from decomposition of the exudation, and narrates himself a case in which pneumopericardium developed in connection with carcinoma of the œsophagus. In this case the most careful search failed to reveal any orifice of communication.

In most cases pneumopericardium results from direct penetration, sometimes as direct as by trauma. Thus Giene reported a case in which paracentesis of the pericardium by a trocar permitted the entrance of air, and Bodenheimer a case in which the pericardium was opened by a gunshot wound.

Numerous cases of perforations from the inside have been reported. Thus Thompson and Walsh saw pneumopericardium caused by opening of the pericardial sac from the œsophagus by a knife that had been swallowed. Chambers and Tütel reported cases of perforations from ulcer of the œsophagus; Saexinger, Rosenstein, and Guttmann cases of perforations from ulcer of the stomach; Graves a case in which the pericardium was opened by an abscess of the liver through agglutination of the stomach and diaphragm; McDowell a case of perforation from a tuberculous cavity in the lungs. Morel-Lavallée saw a case caused by the penetration of the lung and pericardium by the end of a fractured rib. Petit quotes Ledonpacher, Schwartz, and Reyner, who observed cases resulting from violent contusion of the thorax, producing rupture of the lung adherent to the pericardium.

The quantity of air or gas present in the pericardial cavity may vary from a slight, scarcely perceptible amount to such an accumulation as will distend the pericardial sac. Small quantities accumulate at the base of the heart; larger quantities distend the pericardial sac in every direction to such a degree as to escape from an orifice of puncture with an audible hissing or whistling sound. The presence of sulphuretted hydrogen in however small quantity gives the gas a fetid odor. Great accumulations interfere with the action of the heart and displace neighboring organs. In all cases the presence of gas would excite inflammation of the pericardium.

Symptoms.

The symptoms usually set in suddenly with extreme pain, sometimes as severe as that of an angina pectoris, with sensations of anxiety, with dyspnœa, sometimes with cyanosis and sudden collapse. Where the accumulation is slower the signs of pericarditis gradually develop,

and when the quantity becomes extreme, the chest-wall is protruded, at least the intercostal spaces are bulged out in the region of the heart, while the apex is separated from the wall of the thorax. Inspection may show no other signs, but, as a rule, a change of posture of the patient may make the apex stroke again visible or palpable.

Percussion reveals a tympanitic and metallic sound with the dulness of fluid accumulating at the same time. The relative positions of the tympanites and the dulness change with the posture of the patient. The gas floats or rises to the surface and the tympanitic note is therefore perceived in front as the patient lies upon his back, or is observed in the axillary line when the patient lies upon the right side. A change in the line of dulness follows a change of posture from the upright to the semi-recumbent and dorsal posture. Openings of communication between the lungs and the pericardium may develop the cracked-pot sound, which is, however, sometimes heard in pneumopericardium even when the cavity is closed.

Auscultation reveals the friction sound of the associate pericarditis, with the metallic notes which belong to resonating cavities lined by membranes. The auscultatory signs are always the most characteristic, and the most important of these signs is the metallic timbre of the heart sounds, which is usually so pronounced that it may be heard at some distance from the patient. Moreover, the sound of the "falling drop," metallic tinkling, may be present, and any friction sound that may be heard has the same metallic note.

Bricheteau first called attention to the splashing sounds of the water mill, which he described as the shuffling of a water wheel in fluid. All kinds of splashing and gurgling sounds may be heard in different cases—sometimes by the patient himself and sometimes even at some distance from the body. All these churning sounds have the same metallic note.

The action of the heart is usually irregular, the pulse is arhythmic, small, and weak on account of paresis of the heart muscle.

The accumulation may be so great as to lead to dyspnœa from compression of the lungs, especially the larger bronchial tubes, and to cyanosis from compression of the great vessels at the base of the heart. Eisenlohr reported a case of dysphagia from compression of the œsophagus.

Fever may or may not be present. As a rule it is irregular and dependent upon the causative condition or the associate pericarditis. The pulse is usually weak and irregular, partly on account of interference with the action of the heart, and partly on account of the associate conditions.

Diagnosis.

The diagnosis is usually not difficult. The symptoms set in suddenly, with severe pain, sometimes, as stated, with dyspnœa, cyanosis, and collapse. The distention of the pericardium with gas is appreciated by the tympanitic sound furnished to percussion and the interference with the action of the heart as manifested in the condition of the pulse. Inasmuch as fluids are always present, the diagnosis is facilitated by the presence of pyopneumopericardium. The distinctive signs are the metallic note and churning sounds, produced by the action of the heart in a medium of fluid and gas.

The condition might be simulated by an encapsulated pneumothorax. In this affection the normal outlines of the heart could be distinguished by dulness on percussion, while the apex stroke would be neither enfeebled nor dislocated. Change of posture, which would change the percussion sounds, would easily differentiate the conditions. An encapsulated pneumothorax would, of course, be fixed and might constitute a cavity in the region of the heart. Here the sounds of the action of the heart could be separated by having the patient hold his breath. In pneumothorax there is usually amphoric resonance, which is, of course, absent in pneumopericardium. The symptoms are never so intense; the dyspnœa and distress are never so great in an encapsulated pneumothorax. Any confusion caused by a neighboring vomica could be cleared by the same rules.

Accumulations of gas in the stomach often furnish consonant metallic heart sounds. Distention of the stomach belongs to gastrectasia, which in turn would show its cause, gastric catarrh, atonic dyspepsia, carcinoma of the pylorus, etc. In all cases the accumulation of gas would be quickly discharged by the introduction of a stomach tube. The "clock-stroke" consonant sounds in the stomach cease then at once.

Post-mortem accumulations would be differentiated by the absence of the signs of inflammation. Foerster declares that the pericardium is dry in all cases developed after death.

Prognosis.

The course of the disease runs from bad to worse, and usually rapidly because of the nature of the original condition. Of the fourteen cases collected by Friedreich, ten succumbed, some of them in the course of a few days, some of them even in the course of a few hours.

The pneumopericardium which occurs in consequence of outside trauma has, however, a less serious prognosis, and in any case the

result is not necessarily fatal unless such a termination be necessitated from the nature of the original malady. It is certain that gas has been absorbed in the course of two or three days and that patients have recovered from serious attacks of the disease. Aran reported a case in which all the symptoms of pneumopericardium disappeared in the course of a few hours.

The *prognosis* is determined to a large extent by the condition of the heart muscle and the nature of the original malady.

Treatment.

The treatment is addressed chiefly to the support of the heart and relief from pressure by heart tonics, such as digitalis or strophanthus, and by paracentesis pericardii.

Pain may be relieved by the application of cold, or if necessary by the subcutaneous injection of morphine. Dyspnœa is best combated by the diffusible stimulants, caffeine, ether, etc. Conditions of collapse call for stimulation by alcohol and especially for the subcutaneous transfusion of salt solution. Pyopneumopericardium demands incision and drainage.

For the rest, the treatment is directed to the originating malady, ulcer of the stomach, tuberculosis of the lungs, affections of the mediastinum, etc.

DISEASES OF THE HEART.

Congenital Anomalies.

The heart may be absent entirely. During intra-uterine life it has little or nothing to do, as the body of the fœtus is supplied through the umbilical vein by the heart of the mother. Zweifel demonstrated by the spectroscope the presence of oxygen in the blood of the umbilical vein. These cases of acardia belong to the extreme monstrosities and are usually associated with acephalia.

The heart may be too small. In these cases all the parts may be present or certain parts may be absent or but rudimentarily developed. On the other hand, the heart may be too large. Most of these cases are double hearts, the result of the coalescence of twins. One of these hearts is usually more or less imperfectly developed; in rare cases the hearts are separate.

Dislocations.

The heart is sometimes dislocated from its natural position. In certain cases it may lie outside the body entirely, constituting

ectopia cordis. This condition is not incompatible with life for a certain length of time. Lebert speaks of having seen such a case in a new-born child while alive and of having been able to study the movements of the heart very well in it. Martinez described a similar case in a robust, healthy child born at term. The heart deprived of its pericardium was situated in front of the chest, its point being directed forward, its base backward, enveloped by the skin of the chest. The movements of the heart were distinctly visible. The child lived twelve hours.

Defects in the sternum with exposure of the heart are much more frequent. A case of this kind made it possible for Harvey to study the movements of the human heart in life. Breschet made an exhaustive study of displacements of the heart. In certain cases the heart was found to deviate from its normal position without being displaced in its totality. Sometimes the direction of the heart is changed so that the apex points to the right instead of to the left; sometimes the position is vertical.

These displacements are frequently found in association with general transposition of the viscera. In this general transposition the heart lies upon the right side—dextrocardia. The fundus of the stomach lies upon the right side with the spleen, while the pylorus and duodenum lie upon the left. The rest of the intestine is likewise changed. The cæcum lies upon the left, the sigmoid flexure and the rectum upon the right. The ascending and descending colon change names. The liver lies in the left hypochondrium and the right and left lobes change places. The gall bladder, the gall ducts, the ligaments, and fissures show corresponding dislocation. The right kidney lies higher than the left, and the left testicle stands higher than the right. Malformations of individual organs are very rare in typical situs inversus.

The transposition of the viscera is explained by an abnormal habitus with reference to the umbilical vesicle in the embryo. Dareste showed that when a hen's egg is warmed on one side a transposition of the viscera more or less pronounced occurs in the majority of cases. The inversion takes place whenever the embryo does not turn at the right time from the right to the left. The condition is not now so rare as was formerly believed, since the advance of methods of examination disclose the transposition during life. Hitherto the alteration was discovered only upon the post-mortem table. It is a curious fact that the individuals who show the situs inversus are nevertheless always right handed (Rosenbach). The total transposition of the viscera in no way interferes with long life and perfect action of all the organs. Symptoms, when present, are

due to complications or accidental associate malformations. Thus, v. Buhl reported a case of transposition of all the viscera with stenosis of the cone of the pulmonary artery and defect in the septum of the ventricles.

The great majority of cases of so-called transposition are nothing more than dislocations of the heart, which has been crowded over to the right, usually by an extensive pleuritic effusion in the left chest.

Sometimes the heart is situated higher or lower than usual. Breschet divides these changes into the cervical or superior, the abdominal or inferior, and the thoracic or anterior displacements. In most of these cases the heart had escaped from the pericardial sac by a process of so-called hernia of the heart, and in all cases death occurred shortly after birth. Béclard and Breschet described a case of cervical ectopia in which the heart was displaced upward and outward and lay in front of the neck. Three cases of displacement into the abdominal cavity were described by Klein, Sandifort, and Béclard and Breschet. These last-mentioned authors also described a case of displacement upward and outward to constitute a cervical ectopia.

Defects in the Septa.

The *interventricular septum* may be entirely absent, but this anomaly is never observed alone. Where the septum is entirely absent the heart consists of but three cavities, two auricles and one ventricle. Partial defect is more frequent, sometimes as the result of rudimentary development, sometimes as the effect of perforation. Rokitansky described an opening seven or eight millimetres in diameter, perfectly round, and situated exactly in the centre of the septum. Three-fourths of the partial malformations consist in the absence of the posterior part of the interventricular septum.

These defects are distinguished from the defective closure of the *foramen ovale* by the site of the foramen; and all congenital defects should be distinguished from the perforations caused by the rupture of an aneurysm or by acute ulcerating endocarditis. This distinction can be made microscopically by the appearance of the border under the microscope, which would show signs of ruptured aneurysm or of endocarditis. Ford reported a case in which the foramen ovale measured two inches by one and three-fourths inches.

In the complete absence of both septa the heart consists of but two cavities, constituting the condition known as the *cor biloculare*.

Anomalies in the Chambers of the Heart.

The various chambers of the heart may be too large or too small. The right auricle may be dilated to the size of both ventricles. Diminution of an auricle in size is more infrequent and if present is associated with diminution of the corresponding ventricle.

Hypertrophy of the ventricle is more common than dilatation. Concentric hypertrophy of the right ventricle is one of the most frequent anomalies. The walls are thick, the cavity small. The muscular tissue is distinctly enlarged. Sometimes there is evidence of myocarditis in sclerotic tissue, but hypertrophy is the first factor.

The endocardium may present anomalies in thickness, inequalities, vegetations, the signs of existing or pre-existent endocarditis. The pericardium may show the same signs of inflammation in thickenings, opacities, adhesions, etc.

Nodules and free bodies are sometimes found in the cavity of the heart. Parrot described certain of these bodies as hemato-nodules. The hematomata are small, dark, spheroidal bodies, resembling minute coagula, about the size of a millet seed. The nodules are small, fibroid bodies, adherent to the valves. Both the hematomata and the nodules are covered by endocardium. These hemato-nodules are found especially in still-born children, but they seem to be very common during the first fortnight after birth and are sometimes seen up to the age of two or three, exceptionally of seven, years.

Huchard described a number of cases of aberrant chordæ tendineæ—*i.e.*, cases in which the cords, instead of running from the columnæ carneæ to be inserted into a valve, traverse the ventricle directly. Where the cords are so situated as to interpose an obstacle to the current of blood they may give rise to musical murmurs and sometimes to vibratory thrills. In five cases the diagnosis of aberrant cords made in life was substantiated at the autopsy. These anomalies are for the most part congenital, though they sometimes result from sclerotic trabeculæ. They have been found hitherto only in the left chambers of the heart.

Anomalies of the Valves.

Sometimes the valves are more numerous than normal. Supernumerary valves are more frequent at the pulmonary than at the aortic orifice. Four, less frequently five, valves are occasionally found at the orifice of the pulmonary artery. Sometimes the supernumerary valves are of the same size, oftener of less size, than the others.

Babington once found a supernumerary valve above the others. It was very small, but well formed. Variot once found one of the three sigmoid valves higher than the other two (Moussous).

More frequently there is a deficiency in the number of valves. The existence of but two valves is the most common anomaly, and this anomaly is sometimes noticed at the orifice of both aorta and pulmonary artery, coexisting at times with narrowness of the vessel. Fenestration of the valves, most commonly by the existence of perforations or slits near the borders, are sometimes seen. Defects in the auriculo-ventricular valves are much more rare. Leudet (1888) collected all the cases (23) hitherto reported of congenital defect of the tricuspid valve. Abercrombie reported a case of complete obliteration of the right auriculo-ventricular orifice. Similar lesions have been noticed in the valves at the left auriculo-ventricular orifice.

As to the frequency of valve lesions, the conditions prevailing in the fœtus are exactly the reverse of those existing after birth. Thus Rauchfuss found 192 cases of fœtal endocarditis in the right side and but 15 in the left. The cases were selected from many sources. The strain falls upon the right heart in fœtal life.

Anomalies in the Great Vessels.

Anomalies are sometimes seen in the situation of the great vessels. Thus the aorta or pulmonary artery may communicate with each ventricle or with both ventricles. Sometimes the aorta arises from the right ventricle while the pulmonary artery arises from the left ventricle. Anomalies in the further course of the vessel are more infrequent. Sometimes one of the great trunks is lacking or is reduced to a fibrous cord.

The *pulmonary artery* most frequently shows anomalies. Kussmaul bases his classification of congenital defects on malformation of the pulmonary artery and groups all the lesions of the heart about this one. Contraction of the orifice is a common condition. Morgagni reported the first case of congenital malformation of the heart in a stenosis of the pulmonary orifice in a girl sixteen years of age. Sometimes the contraction is caused by the fusion of the pulmonary valves, the coalescence of which may constitute a kind of funnel perforated at the centre and usually bulged upward like a dome into the course of the artery. Sometimes the obliteration is complete and the artery terminates by the side of the heart in a cul-de-sac. More rare is a lesion of the valves which permits regurgitation.

Stenosis of the pulmonary artery is the most frequent of all the malformations of the heart. Kussmaul was able to find records of 90

cases. In these stenoses the foramen ovale was found open in 39 out of 53 cases. The ductus arteriosus was found absent in thirteen per cent. of these cases, a fact which Longstreth thinks tends to support Peacock's view that narrowing of the pulmonary artery is the consequence of the defective development of that branchial arch out of which the ductus arteriosus is formed. The right ventricle is always found hypertrophied in marked degree and the hypertrophy is always associated with dilatation. Sometimes the whole pulmonary artery is preternaturally small. This reduction in the calibre of the tube has long been considered a predisposing cause of tuberculosis of the lungs.

The *ductus arteriosus* sometimes remains permanently open; sometimes it is found dilated; sometimes closed. Persistence of this duct was first recognized by Duroziez and d'Almegro, in 1862. When one end only is closed, the open end is found in connection with the pulmonary artery. The ductus arteriosus and the foramen ovale close normally within four or five weeks after birth.

In cases of closure of the pulmonary artery with persistence of the ductus arteriosus, the blood passes from the aorta through the duct into the branches of the pulmonary artery, which thus become branches of the aorta. When the ductus arteriosus itself is closed or contracted, the bronchial arteries assume the supply for the lungs.

Sometimes the ductus arteriosus is large enough to give passage to a large quill. In these cases the blood passes from the aorta to the pulmonary artery, and the increase of pressure thus induced leads to dilatation with hypertrophy of the right ventricle.

The *aorta* is less frequently affected, but it may be preternaturally large or small. The reduction in size may be effected, as in the case of the pulmonary artery, by fusion of the sigmoid valves. Sometimes the aortic orifice is entirely obliterated. This lesion is rare, but a number of cases have been reported; partial obliteration, stenosis, is more common. Reduction in the size of the aorta itself has been distinctly connected with chlorosis by Virchow, who described it as the aorta chlorotica.

Congenital contraction of the aorta itself is infrequent. Archer reported a case of a heart with a congenital band across the region of the aorta, and Knoevenagel a case of congenital contraction of the whole aortic system with consecutive pronounced hypertrophy of the heart. Rosenbach devoted especial attention to this anomaly because it has clinical importance from the fact that it may cause sudden death, and from the fact that trivial diseases, for instance infections, assume a grave character in the presence of it—that is, unexpected grave symptoms may show themselves on the part of the heart.

Congenital contraction of the aortic system is usually attended by a marked hypertrophy of the left ventricle with contracted and highly tense arteries. But when compensation becomes broken there may be dilatation of the left ventricle and thereupon extreme dilatation of the right half of the heart. Mitral murmurs or the signs of insufficiency of the valves show themselves early. The insufficiency may be due to weakness of the myocardium—that is, to relative insufficiency or to real sclerosis. Systolic murmurs at the orifice are therefore sometimes present. The consecutive hypertrophy and active dilatation of the right ventricle occur early. Orthner distinguished as characteristic of congenital contraction of the aortic system a pulsation in the jugulum arising from the aorta. But this pulsation is often absent where the heart's action is but little excited. Quite characteristic is the continuous subnormal temperature which shows itself during the existence of an infectious disease, which is usually marked by a high temperature. The anomaly is usually associated in women with infantile uterus, undeveloped mammæ, and absence of hair upon the pubes while it is abundant elsewhere (Rosenbach). See also the section on Hypertrophy.

Anomalies of the great veins are much more rare. The superior vena cava sometimes opens into both auricles. Rokitansky mentions a case in which he found two ascending venæ cavæ.

Fallot found as the most frequent lesions, in 39 of 55 cases, causing congenital cyanosis, (1) Contraction or obliteration of the pulmonary artery; (2) Communication between the ventricles; (3) Hypertrophy of the right ventricle; (4) Deviation of the aorta to the right. In seven cases this author found contraction or obliteration of the pulmonary artery with persistence of the foramen ovale. In three cases there was but one arterial trunk, which later separated into the aorta and pulmonary artery, and in but one case was the cyanosis found to be due to persistence of the foramen ovale alone.

Congenital *modifications of the blood* have been described. Vaquez found a hyperglobulism in cyanosis and considered this condition as of importance in the pathogeny of cyanodermia (Moussous).

Etiology.

Some of these changes, permanence of the foramen ovale, etc., are attributed to arrest of development. Peacock divides the anomalies into three classes: First, those which occur early, at the fourth to the sixth week, showing a heart with two or three cavities, with single or imperfectly divided arterial trunk, etc.; Second, arrests at the sixth to the twelfth week, showing imperfect auricular or ventricular

septa, imperfect or misplaced vessels; Third, defects after the twelfth week, showing anomalies of valves, persistence of fœtal openings, etc. Other changes, fusion of valves, hypertrophy, etc., are due to processes of infection and inflammation, *i.e.*, to fœtal endocarditis, arteriosclerosis, etc. Endocarditis in the fœtus is chiefly confined to the right side of the heart. This preference, as is the preference of the left side in adult life, is usually ascribed to the greater work of these sides, respectively in intra- and extra-uterine life. But Rosenbach remarks upon the richness of the blood in oxygen as a chief factor in the remarkable almost exclusive localization of disease before and after birth. This view explains the predisposition of the left side of the heart in extra-uterine life and the indifference of localization in the fœtus, in which both sides of the heart contain about the same amount of arterial blood, or at least show no such difference as after birth.

Heredity plays a rôle in the production of some of these anomalies. Moussous quotes from the Dublin *Medical Press* a case in which three infants born in the same family showed congenital lesions of the heart. Two were born dead; the third, treated several years after birth for laryngitis, showed a musical murmur under the left clavicle, with frequent attacks of cyanosis. Krockez was able to refer a case of congenital contraction of the pulmonary artery with fusion of the valves to syphilis. In this case the mother had had several abortions and showed herself unmistakable signs of syphilis at the time of the birth of the infant. Kuhn attributes fœtal endocarditis to rheumatism of the mother. Dilg reported 15 cases of stenosis at the aortic orifice, 7 of which were evidently due to an endocarditis originating late in fœtal life. The remaining 8 cases were caused by malformation of other parts of the heart.

Symptoms.

Individual and isolated defects, like perforations of the septum, may remain entirely latent. Gage reported a case of interventricular opening in a man who had been in robust health, and who died from an aneurysm of the aorta which burst into the cavity of the right ventricle. Obstruction at an orifice may show no symptoms or but few symptoms so long as compensation is perfect. Even multiple lesions may be marked by but few signs. Thus, Carroll reported a case of congenital malformation of the tricuspid valve with lesions at all four valvular orifices, which survived for twenty years with few heart symptoms during life. But symptoms begin to show themselves so soon as the compensation begins to give way and in correspondence

with the degree of occlusion and of heart failure. The anomalies of the heart are manifold and varied, but the symptoms are singularly uniform, and one of them overshadows in value all the rest.

Cyanosis.—Of all the symptoms of congenital defects of the heart cyanosis is the most obtrusive. Cyanosis, morbus cœruleus, *maladie bleue*, is that bluish discoloration of the skin and mucous membranes caused by the presence of venous blood in the capillaries. The dark-blue blood of the veins, rich in carbonic acid, stands in marked contrast to the bright red arterial blood, rich in oxygen. The color is most distinctly manifest in the most vascular and transparent parts, as in the lips and tongue, face and ears, and in the parts most distant from the heart, as in the extremities. Cyanosis usually shows itself in the first week of life and remits or abates later to show variation in the subsequent history of the case. Thus, cyanosis appeared in Stölker's cases (57 in number), at or soon after birth, 32 times; from the end of the second week to the end of the sixth month, 9 times; from six months to twelve months, 3 times; in the second year once, in the fifth year three times, and in four cases it was absent entirely. Sometimes the cyanosis and other signs of heart affection, palpitation and dyspnœa, make themselves manifest for the first time only after the occurrence of some acute disease, or sometimes, in the presence of compensation, only after the lapse of a long time (a month or even a year) when the compensation begins to give way. Cyanosis is especially pronounced in communication between the auricles, in patulousness of the foramen ovale, and in the presence of great defects in the ventricular wall, so that arterial and venous blood are mixed.

Cyanosis may be present at birth or may, as stated, show itself later. Sometimes it is continuous; sometimes it is present only after effort. In all cases it varies in degree and is especially called out or intensified by effort of any kind. Any muscular movement, especially if prolonged or severe, like the act of crying or coughing, the straining of defecation, an emotional disturbance, which is easily excited in these cases, or a fit of anger, displays or intensifies the color at once. Sometimes the color assumes a darker tint. It may be as brown as the bronze hue of Addison's disease.

The temperature is lowered in the colored parts. It may fall several degrees in the extremities, while the central temperature, as taken in the rectum, remains normal. Cyanosis of the face, hands, and feet is often accompanied with ectasia of the superficial veins (Lebert).

With the appearance of the discoloration may be seen other signs of interference of the circulation. The act of respiration may become

panting. There is sometimes severe palpitation, attended in older children with a sense of anxiety. Bad cases may be attended with epilepsy.

Certain cases show an extreme vaulting (*voussure*) of the chest in the precordial region. The heart wall itself under great hypertrophy is usually visibly shaken by the shock. Murmurs are usually distinguished by their great intensity and wide dissemination, so that they may be appreciated even when the ear is removed from the stethoscope.

As already stated, deformities of other parts of the body are frequently present. Blue babies are rarely perfectly developed. The thorax is contracted, the lungs are small, the ends of the fingers and toes are clubbed, dentition is delayed and is irregular. The whole process of hematosis is naturally defective. Hendly reported a congenital malformation of the heart with the appearance of six incisor teeth of the upper jaw; Orth a case in connection with imperforations and hypospadias; Barbillon the case of an infant affected with cyanosis from perforation of the interventricular septum and contraction of the pulmonary artery, which showed also asymmetry of the cranium in the parietal and frontal regions, atrophy of the lobe of the ear, contraction of the external auditory canal, and atrophy of the mastoid and temporal processes. Rokitansky noticed cleft palate, and Kane spina bifida, in connection with cyanosis. In a case of cyanosis reported by Monnier, the child was an idiot and showed deformities of the jaw and foot. Church reported an ichthyosis in connection with a congenital malformation of the aorta, Guyon a case of hysteria, and Eichhorst a case of deaf-mutism in association with congenital defects of the heart.

Diagnosis.

Cyanosis alone does not suffice for the diagnosis of a congenital lesion of the heart. Every child is born more or less cyanosed from compression upon the umbilical cord or somewhat premature detachment of the placenta. Any doubt whether a cyanosis depends upon congenital defect of the heart or mere interference with respiration is determined at once when the child breathes, as cyanosis from defective aëration of the blood, as from a too long-continued pressure upon the umbilical cord, entirely disappears with the establishment of the act of breathing. In fact, the blue color disappears shortly after the child cries. Should the cyanosis persist, it depends upon either malformation of the heart or occlusion of the lungs, as by atelectasis. Cyanosis due to cardiac defects shows regular but hurried respiration; cyanosis due to atelec-

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tasis shows defective expansion of the chest with sinking of the jugulum, epigastrium, and intercostal spaces. But where the cyanosis is so extreme that convulsions ensue, the diagnosis may be established only upon the post-mortem table.

It must be remembered, also, that there are cases of cyanosis, congenital or acquired in early youth in which any defect of the heart, endocarditis, or arrested development, may be excluded, and the blue color may be due to insufficiency of the lungs from emphysema and subsequent bronchial catarrh. Rosenbach is sure that certain cases are due to abnormalities in the condition of the lung capillaries or to anomalies in the gas interchange on account of affection of the lung endothelium without any implication of the heart. These cases may be attended with cyanosis of extreme degree, so that cyanosis, congenital or acquired early, may not be regarded as absolutely characteristic of failures of development in the heart.

It must be remembered, further, that the absence of cyanosis does not exclude congenital defects of the heart. Lebert maintains, indeed, that complete absence, late appearance, or slight development is by no means rare. Landouzy reported a case of large communication between the auricles without cyanosis, Farquhar a case of open foramen ovale not attended with cyanosis until two or three days previous to death, and MacGibbon a case of mitral regurgitation with great hypertrophy of the heart, systolic murmur audible at a little distance from the surface, open foramen ovale, and entire absence of cyanosis.

The fact that, notwithstanding septum defect and other malformations, which usually produce the characteristic symptoms of morbus cœruleus, cyanosis may be absent, would seem to lend support to the view of Cruveilhier, who derived the cyanosis from venous stasis and defective absorption of oxygen—that is, from a disturbance of chemical function and not from a mixture of arterial and venous blood. We might venture to enlarge this view with the statement that a number of cases of anomalies in development of the heart are associated with anomalies in development of the lung capillaries, especially in the lung tissue, so that the gas interchange may be not a mere mechanical effect but a direct biological process. These anomalies may interfere with the specific secretory activity of the lungs (Rosenbach).

We are therefore justified in attributing symptoms to congenital defects or to fœtal endocarditis only when they show themselves at the earliest age. These symptoms are, as stated, protrusion of the chest, abnormal action of the heart, great dyspnœa, palpable tremor in the precordial region, abnormal sounds, etc., and these symptoms assume a special prominence when attended with a pro-

nounced cyanosis, and when the development of the whole body is distinctly arrested.

Sometimes, but by no means always, a diagnosis of the particular defect may be determined.

Stenosis of the pulmonary artery reveals itself by a systolic murmur heard in the greatest intensity at the base near and to the left of the sternum, in the second intercostal space, sometimes also by a frémissement cataire. The murmur is usually so intense as to be propagated a considerable distance, so that it may be perceived distinctly under the clavicle, in the back, between the scapulæ. The right heart is hypertrophied so that dulness reaches up to and sometimes beyond the right border of the sternum. The heart beat is forcible. The apex may be somewhat dislocated to the left. Attacks of palpitation are common; they are usually accompanied by dyspnœa with cyanosis. Any associate insufficiency of the pulmonary valves is distinguished by the presence of a soft, blowing murmur, just pre-systolic or synchronous with the second sound.

Congenital contraction of the aorta is manifest by a bruit, sometimes accompanied by a frémissement cataire, heard in the greatest intensity at the base near and to the right of the sternum and synchronous with the first sound of the heart, by hypertrophy of the left ventricle, with increase in the vertical dulness, and by a small, hard pulse. A congenital or acquired contraction of the aortic system may only be accepted with certainty when it is coincident with secondary changes in the heart, hypertrophy and dilatation.

Contraction of the part of the aorta which lies between the origin of the left subclavian artery and the orifice of the ductus arteriosus, the so-called isthmus of the aorta, with obliteration of the duct, occurs most frequently in the male sex. The arteries which ascend from the arch of the aorta to the upper half of the body are now remarkably large, while the arteries of the lower half of the body are correspondingly small. The pulse in the abdominal and crural arteries is small, almost imperceptible and is retarded. In consequence of the grave disturbance in the circulation the left ventricle undergoes hypertrophy, and there develops between the upper and lower half of the body a collateral circulation which is so marked as to be both visible and palpable. The visible evidence of this anastomosis is seen in the great swelling and pulsation of communicating vessels, especially in the region of the inner border of the shoulder and the anterior wall of the chest. The arch of the aorta, subjected to the pressure of the stronger blood wave from hypertrophy of the left ventricle, is gradually dilated and the pulsation of it may be felt in the neck behind the manubrium sterni (Leube).

Persistence of the ductus arteriosus is indicated by the presence of a murmur, which is heard best at the back of the thorax at the left of the spinal column on a level with the third and fourth dorsal vertebræ. The murmur is intensified by inspiration and diminished in expiration. On holding the breath it shows uniform intensity. The pulse shows arythmia; a series of four or five strong pulsations are followed by feeble pulsations. The inequality stands in connection with respiration. Gerhardt noticed that these infants are lightly cyanosed or show a waxy pallor and are the subjects of dyspnœa and bronchial catarrh.

Defective closure of the interauricular septum—that is, persistence of the foramen ovale—is indicated, according to Sansom, by cyanosis accompanied by systolic and pre-systolic murmurs, most distinct at the level of the third and fourth intercostal cartilages.

Perforation of the interventricular septum is indicated, according to Roger, by the presence of a bruit heard in maximum intensity in the upper third of the precordial region, about the third intercostal space. The bruit is fixed, that is, is not propagated into the vessels, and is unaffected by the movements of respiration or by the posture of the patient. It is sometimes so intense as to be heard in the back. Cyanosis, which is absent at first, may appear later at a more advanced age, particularly in the presence of any lung disease, especially of tuberculosis.

Prognosis.

Cyanotic children succumb readily to broncho-pneumonia, atelectasis, and hemorrhage. There is also a tendency to gangrene. Bouillaud reported the death of a cyanotic infant from hemorrhage of the gums. Many cases succumb to tuberculosis. Thus Louis reported 3 deaths from tuberculosis in 7 cases of cyanosis, Gintrac 7 deaths in 16 cases.

In a general way it may be said that eight per cent. of the cases die within the first week; thirty-six per cent. within the first year. According to Longstreth, one-fourth of those who die in infancy succumb in paroxysms of dyspnœa, one-fourth succumb to some intercurrent acute disease, and one-half of the fatal cases perish in convulsions; in all cases the cyanosis deepens at the close.

The prognosis depends, however, in large degree upon the character of the lesion. Thus cases of complete obliteration of the aortic orifice may not survive longer than seven to nine days, while cases of mere contraction may live from five to nineteen years. Cases of complete obliteration of the pulmonary artery usually succumb in a few days, but exceptionally cases have been recorded which have

lived five to eight months. Malformations of the septa are often associated with other defects, so that cases may survive for a few weeks to a number of years—in fact, individual cases have reached maturity, and malformations confined to the interauricular septum may not be incompatible with a longer life, up to the age of forty even to fifty years. Cases of transposition of the great vessels may live from two to five months. Stenosis of the pulmonary artery with persistence of the foramen ovale, which constitutes the most frequent lesion, cuts life short after periods varying from one to forty years.

Lesions are often disclosed at an autopsy which surprise the pathologist by the length of life. Thus Mollwo reported a case of central origin of the aorta, defect in the septum of the ventricles, deep origin of the pulmonary artery, beginning at first within the musculature, with the preservation of perfect health up to the eighth year of life. Walsham reported a case of unobliterated ductus arteriosus without other malformation of the heart and great vessels in a man aged forty-seven, Baldwin a case of dropsy involving only the lower extremities and half of the trunk from malformation of the heart in an adult, and Blake a case of open foramen ovale in the dissection of a soldier. Finally Longstreth quotes a case of closure of the pulmonary artery with persistence of the ductus arteriosus and substitution of the pulmonary by the bronchial arteries in an individual aged thirty-seven years.

Treatment.

As many anomalies are not incompatible with life, an effort should be made to fortify the heart that it may compensate the defect.

The still-born child must be treated according to the usual methods, the best of which is, after the removal of mucus from the throat, the Schultze plan of swinging by the feet. In the experiments of Zunze and Strassmann still-born children were put under the same conditions as in the vagina, and the various methods of artificial respiration were employed to inflate the lungs. In this way it was seen that the lungs of a fœtus were filled with air under the Schultze method. Sometimes it is advisable to inflate the lungs by means of a catheter introduced into the trachea. After the lungs once contain air the Sylvester method of compression of the thorax is the best in sustaining respiration.

So soon as it is determined that the cyanosis depends upon organic disease, the child should be laid upon its right side to counteract, at least to some extent, by gravity a possible defect in the septum of the auricles. In all cases, premising that respiration has been established, the body should be enveloped in hot blankets and

placed near a fire, or, as soon as the necessary service has been rendered, in bed with the mother.

The heart is now best strengthened by keeping the surface of the body warm, while the lungs are fed with fresh air, by attention to the digestive tract and by protection of the child, best by isolation, from exposure to the infections of infancy, especially from whooping cough and measles. Sojourn in a mild, warm climate, with proper regard for the clothing, best protects the heart against the diseases which result from or are aggravated by vicissitudes in the weather. In the course of time the heart may be toned by graded exercise, which must be supervised with the utmost solicitude.

Especial attention must be paid to the choice of a profession. Matrimony should be forbidden to both sexes, but especially to women on account of the dangers of pregnancy and parturition, which become extreme in these cases. The patient is to be early taught the virtue of self-control, and the necessity of cultivating a cheerful temperament.

Attacks of palpitation and cyanosis may be relieved by the application of cold to the chest. Especially may be recommended in these cases the wearing of the hollow tin shield filled with cold water. Nervous excitement may be subdued by the use of the bromides. The heart may be sustained by the exhibition from time to time of strychnine, the tincture of nux vomica, the tincture of digitalis, etc. Constipation may be avoided by the use of mineral waters, Carlsbad salts, or preferably by some of the vegetable laxatives, rhubarb, cascara, aloes. Cases of threatened heart failure may call for the use of the stronger stimulants, alcohol, ether, caffeine, according to the principles detailed elsewhere in the management of heart failure.

The diet is to be regulated to a nicety as every imprudence is quickly punished. Frequent bathing is of great value in supporting the heart through the nerves of the skin, and fresh air in abundance is a necessity in the manufacture of blood and in the feeding of the tissues.

In this way life may be prolonged, even in the face of congenital defects, to fifty years, so that prudent individuals thus afflicted may survive by decades of years the more fortunate but more reckless possessors of perfect hearts.

Atrophy.

The heart varies in size within certain limits, but is roughly estimated to be about the size of the closed fist of the individual. Some individuals have naturally large hearts, some have small hearts.

These anomalies are congenital and usually coincide with excesses or deficiencies in the whole arterial system. Congenital atrophy of the heart goes along with a general arrest of development. The condition fortunately is not propagated, as it is usually associated with atrophy of the genital organs. More commonly, however, the conditions are acquired. Hard workers have absolutely larger hearts; the obese, aside from deposits of fat, have relatively smaller hearts. As a rule, on account of arteriosclerotic processes the heart enlarges, but in exceptional cases it shows atrophy with the general marasmus of old age. This atrophy of old age, when it occurs, is a part of the process of general senile involution.

The heart of an adult man weighs, on the average, ten to twelve ounces; of a woman, eight to ten ounces. In disease processes the heart usually undergoes hypertrophy and the enlargement is compensatory to overcome obstacles to the circulation of blood. When in the course of time the heart muscle has exhausted itself in this effort, it suffers degeneration of various kinds. With the substitution of muscular by fatty or other inert tissue, the heart loses its power of resistance. The chambers of the heart expand, and the heart is said to suffer dilatation. So that in the subsequent course of the disease, while the heart muscle, being substituted by fat, is actually lost, the heart itself is not, as a rule, reduced in size but remains larger than normal.

Nevertheless in certain cases there may be absolute reduction in volume and sometimes, especially in the course of wasting diseases, the heart undergoes actual atrophy.

The atrophy may be general and concern the whole heart, or local and affect only parts of the heart. The process may be limited to the papillary muscles. Morgagni, Burns, and Chomel reported cases in which the heart was reduced to one-half its normal volume. Bellingham reported a case of congenital smallness of the heart. Other cases are recorded in the section on Congenital Defects. Bouillaud once found a diminution in weight to 135 grammes (4 ounces). Farell saw a heart which weighed only 3½ ounces, and Bramwell cited a case in which the heart of a woman who died of cancer of the stomach weighed only 2 ounces, 2 drachms, and 11 grains. Finally, Quain reported a case in which the heart weighed but 1 ounce and 4 drachms, though in this case the patient was a girl but fourteen years of age.

Cases of partial atrophy may concern, as stated, only one of the chambers of the heart or any part of the wall of the heart. Sometimes one ventricle is reduced to appear as a mere appendix of the other, or the walls may be atrophied in places, as under the influence

of aneurysm and abscess, so as to suffer actual rupture. Atrophy of individual chambers in the heart is usually only relative; thus a normal ventricle appears atrophied by the side of a hypertrophied ventricle. But sometimes one ventricle undergoes absolute atrophy. Féréol reported a cardiac atrophy consecutive to an old endo-pericarditis, limited principally to the right ventricle. Atrophy of the walls of the heart is usually the result of an arteriosclerotic process.

General atrophy is induced especially in connection with wasting diseases—thus in marasmus from any cause. Stokes long ago described the atrophy of the heart in phthisis; Tigri noticed the wasting of the heart in the insane, and Bramwell observed the atrophy which the heart undergoes in amyloid degeneration of the kidneys. Atrophy of the heart occurs, as a rule, in cancer, diabetes, Addison's disease, etc. Sometimes the heart wastes from local cause. Thus, Barlow, Chevers, and Walshe recorded cases of atrophy in the course of pericarditis, and Smith and Porta in connection with calcification of the pericardium.

In this process of atrophy the consistence of the heart varies. It is usually lax and flabby from the predominance of fatty degeneration. The myocardium may be lighter, assuming something of a yellow color, or darker with a brownish hue from the accumulation of pigment matter. The muscular substance may be reduced to such an extent as to throw the visceral pericardium into folds, and such a shrunken brown heart, covered by wrinkled pericardium, has been likened to a "withered apple." On the other hand, localized atrophy may show much firmer consistence. This process depends, as stated, upon arteriosclerosis; and the atrophied regions may consist largely of cicatricial tissue. Friedreich described a general sclerotic atrophy, and partial cicatricial degeneration has been studied as a special form of myocarditis, as a cirrhosis of the heart, usually, however, in connection with a general hypertrophy.

Symptoms.

Atrophy reveals itself rather by weakness in the action of the heart than by any gross or obtrusive physical signs. The weakness may be apparent in the diminished impact and feeble stroke, in the weakness of the pulse, and especially in the defective blood supply to the brain, the lungs, and the kidneys. The weak heart is found in association also with weakness and degradation of other organs and of the whole body. Tuberculosis, cancer, diabetes, and any chronic affection marked by marasmus, naturally wastes the heart with other structures, and an atrophied heart may be assumed in most of these

conditions. Feebleness of effort and early exhaustion indicate weakness in the action of the heart. Attacks of fainting and syncope, confusion of mind, spots before the eyes, ringing in the ears, show the defective blood supply to the brain. The supervention of short breathing, with palpitation of the heart, distress in the precordial region are signs of defective blood supply to the lungs; and a scanty excretion of urine of dark color, with high specific gravity and heavy sediment, shows the diminution and stasis of blood in the kidneys. All these signs point, however, only to weakness in the action of the heart and are, therefore, more frequently encountered in conditions of dilatation which follow hypertrophy. They indicate atrophy of the heart only when found in association with diseases which waste the substance of the heart or in which they may be corroborated by physical signs.

The physical signs themselves are few. Inspection may reveal a diminished or absent impact and apex stroke, conditions which are more distinctly perceived by palpation. These features have more importance from the fact that atrophy of the heart is usually associated with more or less general emaciation, which makes the movements of the heart more manifest to the sight and touch. Percussion shows the diminished area of dulness both absolute and relative. Dulness may be so slight as to be scarcely perceptible at all. It is the dulness which chiefly distinguishes atrophy from dilatation, as with the same signs of weakness dilatation shows increase in percussion dulness. But in all cases allowance must be made for the condition of the lungs. An hypertrophied heart when overlapped by the lung may show but little dulness, and any decided emphysema may suffice to obliterate all trace of dulness in a heart of normal size. The character of the sounds of the heart as revealed to auscultation will depend altogether upon the condition of the heart muscle. Where the heart muscle is sufficiently strong the sounds may be clear and distinct, the diminution in force of the smaller heart being made up for to considerable degree by the thinness of the parietes of the chest. Where the heart muscle itself is weak the sounds will be correspondingly feeble. In these cases the first sound, that of ventricle contraction, may be muffled or absent altogether, while the second sound remains perfectly distinct. In fact, in certain wasting diseases the second sound is even more distinct than normal, and in tuberculosis the accentuation of the pulmonic valve sound, on account of adhesions and superficial position and on account of the better conductivity of solidified lung, is a sign of actual value in the diagnosis of the disease.

The *diagnosis* is determined, therefore, by the association of these

signs: (1) By the genetic relationship—that is, by the existence of some wasting disease; (2) by the signs of defective blood supply to the various organs of the body; (3) by the physical signs on the part of the heart, chiefly by the diminution in the area of percussion dulness.

Prognosis.

The prognosis depends altogether upon the originating malady. The atrophy which belongs to tuberculosis, cancer, diabetes, or Addison's disease, is a grave sign. The condition of the heart is an actual criterion of the imminence or degree of danger. Thus a small, weak heart makes the prognosis bad in tuberculosis, and atrophy of the heart in any wasting disease of progressive character is a *signum mali ominis;* but the atrophy which occurs in the course of any protracted infection, or which sets in early in the history of tuberculosis, may be relieved entirely in the course of time with convalescence and recovery from these diseases. This is especially true of the partial atrophy which develops in long-continued cases of typhoid fever and rheumatism, and especially in the wasting processes which sometimes occur early in tuberculosis and which are checked by change of climate or by the timely and properly graded use of tuberculin.

Treatment.

The treatment of atrophy of the heart is a matter of address to the cause of the condition with sustentation of the general strength, and stimulation of the heart itself, especially by strychnine, roborant diet, pleasure-exciting emotions, sunshine, and fresh air.

Hypertrophy and Dilatation.

The recognition of hypertrophy of the heart during life dates from the discovery of Auenbrugger, whose "Inventum Novum," published in 1761, made it possible by percussion to outline the limits of the heart. This communication, as stated elsewhere, found no general adoption until the forgotten work of Auenbrugger, written in Latin, was resuscitated in French dress by Corvisart in 1811. By the use of this means of study Corvisart was able, as he declared, to diagnosticate hydrops pericardii and aneurysm of the heart, by which was understood what we now know as dilatation. Corvisart was then able to recognize, as Auenbrugger had before him, hypertrophies and dilatations. By the time of Stokes (1850) it began to be possible to separate these conditions. Every clinician became familiar with the cases of hypertrophy which ensued in the course of

valve lesions, and with the dilatation which was consecutive to the hypertrophy. But as long ago as 1827 Richard Bright had called attention to an enlargement of the heart which occurred in the course of disease of the kidneys, and as early as 1856 Traube had devoted special attention to this subject and had pointed to enlargement of the heart as a sign of Bright's disease. The study of the cause of this relationship, or of the dependence of hypertrophy of the heart upon occlusion in the kidneys, led directly to the recognition of arteriosclerosis, or, as it was called later by the English observers, Gull and Sutton, arterio-capillary fibrosis, as a condition which puts obstacles in the way of the circulation and leads as a necessary consequence to hypertrophy of the heart. As it was soon seen, however, that either condition may exist without the other, a marked hypertrophy without arteriosclerosis, or an exquisite arteriosclerosis without any hypertrophy, Traube came to the conclusion that one condition did not necessarily cause the other but that both might be effects of a common cause.

Hypertrophy of the heart was noticed also in the course of pregnancy by Larcher in 1859, and the condition seemed to be established by the French observers, especially by Ménière and Jacquemier. But Gerhardt threw doubt upon the existence of hypertrophy in this condition by attributing the increase in dulness to the higher stand of the diaphragm and protrusion of the heart against the wall of the chest. This objection seemed to have been verified by Löhlein, so that there was a general inclination to surrender the view until it met with readvocacy by Bollinger a few years ago.

Since Bauer (1860) reported a number of cases of simple hypertrophy, and Da Costa, in 1867, noticed the effects of irritation of the heart in army life, the fact that the heart may undergo hypertrophy independent of any obstacle to the circulation in the heart itself—that is, independent of any valve lesion—has been universally established.

These cases of enlargement of the heart in which the increase in volume cannot be attributed to any obstacle in the heart itself are rather inappropriately distinguished as idiopathic hypertrophies. The term is a misnomer, from the fact that in most cases the cause of the condition can be determined as something which raises the blood pressure and in this way opposes the work of the heart. In some cases, it is true, the cause of hypertrophy remains concealed; these cases might be said to be cryptogenetic; but in no case may the hypertrophy be considered as essentially autochthonous or idiopathic; these are terms which should now be abolished in the etiology of disease. The term "peripheral hypertrophy" suggested by certain English writers is too inexact for adoption in our day. Un-

fortunately, the term idiopathic hypertrophy has become so thoroughly incorporated into the history of medicine to express an enlargement independent of disease of the heart itself that use must still be made of it in describing the condition.

As observations multiply it is seen that so-called idiopathic hypertrophy, so far from being rare, is of very common occurrence. A number of cases were reported in the beginning of the seventh decade of this century by Meyer, Da Costa, Clifford Albutt, and Thurn. Fraentzel reported 19 cases of dilatation and hypertrophy which occurred in consequence of the strain of the campaigns of 1871. Schmidbauer and Athmann found the affection very frequent in Munich. Bollinger attributed the frequency of this hypertrophy of the heart in Munich to the plethora caused by the excessive ingestion of beer. In fact, the condition has now become so frequent that while in older text-books this hypertrophy was given but scant description, it is now considered at even greater length than the hypertrophy from valvular disease.

The heart of the adult weighs, on the average, from 300 to 400 grammes. It is said to be hypertrophied when it weighs 400 to 450 grammes, and cases have been recorded in which the heart has weighed 1,000, 1,250, and even 1,700 grammes. These figures translated read: The heart of a man weighs on the average about 9 ounces, of a woman 8 ounces. Anything over 12 ounces in a man and over 10 ounces in a woman constitutes hypertrophy. Usually a hypertrophied heart weighs from 15 to 25 ounces. Metcalfe reported a case of hypertrophied heart weighing 42 ounces, King a case in which the heart weighed 44½ ounces, Gibb a case in which the heart weighed 46½ ounces, Alonzo Clark a heart which weighed 57 ounces, Beverley Robinson a heart which weighed 53 ounces, and Stokes a heart which weighed 64 ounces.

The thickness of the wall of the left ventricle in the adult is from 9 to 12 mm., of the right ventricle from 4 to 5 mm., so that a thickness of 15 to 16 mm. in the wall of the left ventricle, and of 7 to 8 mm. in the wall of the right ventricle, would constitute an hypertrophy. Anything above 4 to 5 mm. in thickness constitutes a considerable hypertrophy in the wall of the auricles. The microscopical measurements by Hepp of normal and hypertrophied muscular fibres seemed to prove that in the process of hypertrophy the fibres are actually increased in number, and that the increase in volume is due chiefly to increase in number and not to increase in size. But upon this point authors differ. The older observers, Vogel, Kölliker, Förster, Lebert, Hyrtl, Rokitansky, believed in the augmentation in the number of fibres—that is, they attributed the

hypertrophy to a hyperplasia. Heschl believed in the new formation of fibres. Robin, Wedel, Becquerel, Paget, and Zenker showed that the fibres of the heart actually increased in thickness in the process of hypertrophy. Zielonko separated the fibres in four different parts of the heart and made comparative studies, claiming that the fibres in the hypertrophied parts were but very little larger, were in fact almost the same size as the normal fibres. Goldenberg his investigations found the heart fibres decidedly increased in thickness and maintains that the estimate of Hepp is too small, because it was made upon the heart of an emaciated woman. Audeoud and Descombes, in reviewing these statements, conclude that the chief element in hypertrophy is increase in the volume of pre-existent fibres; the hypertrophy chiefly concerns the interstitial tissue, and in this interstitial hyperplasia the muscular tissue is destroyed rather than increased, as only vestiges of it can be found in the form of atrophied fibres.

The heart may be hypertrophied in its entirety, that is, in all its parts, or only in certain parts. Where obstruction is offered in the course of the general circulation it is the left ventricle which undergoes hypertrophy; when the obstruction is in the lungs, whether direct or indirect (as in the case of insufficiency of the mitral valves), the right ventricle enlarges. The auricles enlarge in the face of a stenosis at the auriculo-ventricular orifices. Thus hypertrophy may be general or partial. Amburger reported the case of a colossal heart in which the enlargement affected the right ventricle exclusively, the left ventricle being attached to it like an appendix.

Hypertrophies of the heart may be divided into the following groups:

(1) Hypertrophies due to obstacle in the heart itself. This condition is typically represented in lesions of the valves.

(2) Hypertrophies due to increased resistance in the vascular system, as, for example, in arteriosclerosis.

(3) Hypertrophies due to diseases of the heart muscle itself, from infection, over-strain, degeneration, etc.

(4) Hypertrophies due to affection of the nervous system.

I. Hypertrophy from Valve Lesion.

This form of hypertrophy is the overgrowth of tissue which occurs in any organ called upon to do extra work. Forget speaks of the hypertrophy due to an obstacle as a "retro-hypertrophy" and compares it to that which occurs in the bladder in the face of an obstacle in the urethra, that of the stomach which occurs when there is an

obstacle at the pylorus, or in the œsophagus or intestine behind strictures. Bean calls this hypertrophy providential. Hypertrophy in the heart is most commonly seen in the right ventricle in the presence of insufficiency of the mitral valves. Endocarditis is the most common cause of valve disease, and endocarditis attacks by preference

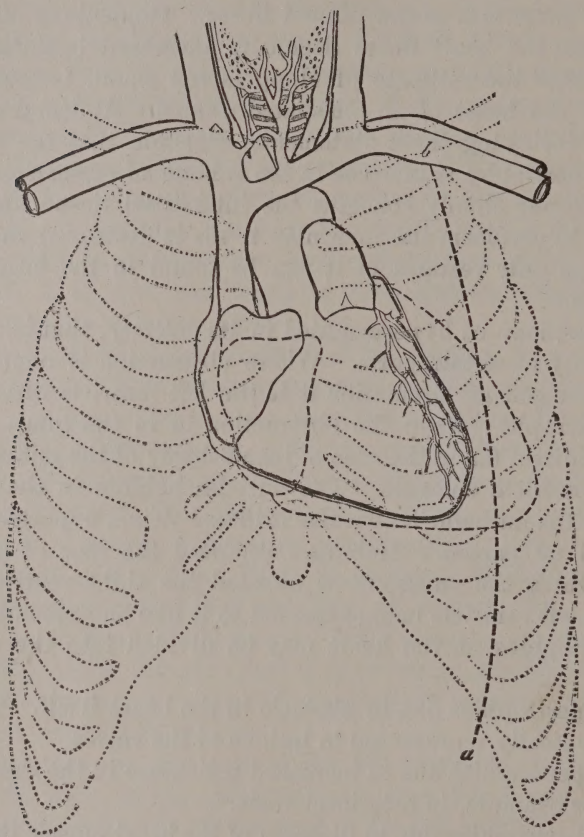

FIG. 3.—Hypertrophy (or Dilatation) of the Left Ventricle. Displacement of the apex beyond the mammillary line (*a*, *b*).

the mitral valve, affecting it in such a way as to render it incompetent, so that the blood which should all escape from the left ventricle through the aorta, regurgitates, some of it, through the left auriculo-ventricular orifice into the left auricle. Under this increase of pressure the left auricle is distended and for a short time overcomes the difficulty by hypertrophy of its structure. But the auricle soon yields and is no longer able thoroughly to expel its contents. In this way the blood pressure is increased in the whole domain of the

pulmonic veins, in the lung capillaries and in the pulmonary artery. Under this increase of pressure the right ventricle enlarges and by reason of the increase in number and size of its muscular fibres is able for a long time to overcome the obstacle in the pulmonary artery. Such an hypertrophy is said to be "compensatory," and so

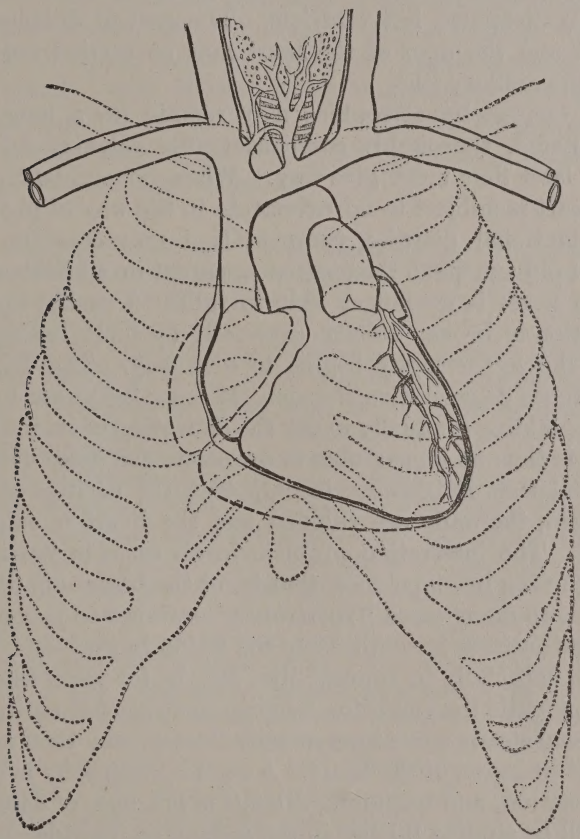

FIG. 4.—Hypertrophy (or Dilatation) of the Right Ventricle; Pulsation at the Epigastrium.

long as it continues intact it entirely counteracts the effect of the lesion in the mitral valves.

Under this hypertrophy the right ventricle becomes as thick and strong as the left; sometimes the right ventricle exceeds the left, so that the normal relation to the heart is changed. In this way the right ventricle forms the apex of the heart, and as the right ventricle reaches over into the right side of the chest, the increase in dulness is apparent at and beyond the right border of the sternum.

When the obstacle to the circulation is offered at the aortic valve, it is the left ventricle which undergoes hypertrophy. When the lesion is of such character as to permit regurgitation, the left ventricle enlarges often to an enormous extent, to constitute the condition which is sometimes distinguished as the *cor bovinum*. The whole chest wall may be agitated under the powerful contractions of the enormously distended left ventricle. The area of dulness increases to the left and the apex of the heart may be carried outward, even as far as the axillary line.

These various hypertrophies may remain for a long time, and may, as stated, thoroughly compensate the valvular defects. But sooner or later they must give way. When no accident supervenes and the heart is subject to no overstrain in the way of physical exertion or emotional distress, compensation may continue until the changes of old age make themselves apparent in additional demands which the heart is no longer able to fulfil. Usually, however, the compensation is broken sooner, especially by some intercurrent disease and the hypertrophy begins to give way. The heart muscle now undergoes degenerative change. It loses its power to overcome obstacles, yielding gradually under the distention of its walls. When the heart is thus no longer able to overcome the defects or obstacles in the circulation, it becomes incompetent, the cavities of the heart are enlarged, the walls are thinned and the heart is said to suffer dilatation. The interesting problem in all cases is the recognition of the beginning incompetence, that is, of the beginning dilatation of the heart. In many cases hypertrophy continues so pronounced and complete as entirely to compensate the lesion in the heart, so that an individual, especially in younger life, may be for years the subject of valve lesion while the condition remains unsuspected until, as stated, some unusual strain, some intercurrent disease, may then make manifest the latent lesion of the heart. A good hypertrophy is a guaranty of compensation, and beginning dilatation is a sign of danger. This subject will be studied further in the section on Valvular Disease.

II. Hypertrophy due to Increased Resistance in the Vascular System.

(*a*) *Congenital Contraction of the Aortic System.*—The size of the arteries varies in different individuals. The vascular system is usually apportioned to the size of the individual and the wants of the tissues. Moreover, as the heart is a development of the vascular system, this organ corresponds in size and power to the demands of the tissues and the size of the vessels. But sometimes this proportion is disturbed. The disturbance occurs most frequently as the

result of disease. The contraction which results from the processes of arteriosclerosis constitutes a most important chapter in the history of hypertrophy. More rarely the contraction of the arterial system is congenital. In any case the reduction in size of the vascular system would have the same effect. It would oppose an obstacle to the circulation of the blood and thus lead to hypertrophy of the heart.

Congenital Contraction.—Morgagni, in his familiar work on the "Seats and Causes of Disease," described exquisite cases of narrowness, "angustia," and Meckel (1755) observed hypertrophy of the heart in consequence of the defect. Virchow (1872) devoted especial attention to the study of the subject and associated congenital narrowness of the aorta with chlorosis.

Fraentzel reported a case which was strikingly illustrative of the condition, where the diagnosis was made in life and where the hypertrophy of the heart could be followed in its gradual development. A robust, vigorous-looking man, aged thirty-four, by occupation a gardener, entered the Charité to be relieved of dyspnœa, palpitation, and cough with bloody sputum. The dropsy was confined to the legs. There was some cyanosis of the face, and such severe dyspnœa as to necessitate the upright posture. The patient stated that he had been compelled to give up military service and to desist from his work on account of the palpitation and dyspnœa which supervened upon every effort. The heart was found in a state of marked hypertrophy. The apex struck in the sixth intercostal space, two and one-half inches outside of the left mammillary line. The impact of the heart was as great as that which occurs in insufficiency of the aortic valves. The dulness extended from the apex stroke to the right border of the sternum. The sounds of the heart were intense and clear. The radial artery was narrow, tense, and thick-walled, as were also the carotids and crurals. The condition had attracted no particular attention in youth, but as age advanced the patient found himself incapable of military duty and of hard work in civil life. After treatment by rest and digitalis the patient left the hospital, but was compelled to return in the course of a few months. The symptoms were now all more severe. The dyspnœa was more continuous, the hydrops more extensive. The brown-red sputum showed the existence of hemorrhagic infarction of the lungs. At the autopsy, which occurred in the course of a month, the heart was found enormously dilated in every direction, but the valves showed no changes whatever. The papillary muscles were elongated and thickened; the coronary arteries were clear, but the aorta from its beginning was unusually narrow. There was diffuse sclerosis of high degree, with gelatinous plaques in the intima. This condition was marked in the ascending aorta, in the arch, and in the abdominal aorta, where the sclerotic process ceased rather suddenly. Enormous hemorrhagic processes together with an extensive œdema were observed in the lungs. The diagnosis which had been made in life was thus established in every particular.

Penzoldt reported two cases of the same kind. One was that of a young girl who succumbed to heart disease with œdema. In this case there was found, besides a slight mitral insufficiency which could not account for the symptoms, a considerable dilatation and hypertrophy of the left ventricle and a distinct contraction of the aorta. The other was that of a young robust soldier, who had to give up his post in a cavalry regiment on account of heart failure from dilatation. Here, too, there was found abnormal narrowness of the aorta. In the case of the soldier the condition had been aggravated by the ingestion of beer, but the dangerous symptoms had supervened only after the strain of military service. This subject has also been considered in the section on Congenital Anomalies.

(*b*) *Hypertrophy from Arteriosclerosis.*—This form of hypertrophy finds its most frequent illlustration in the so-called physiological changes of old age and in the enlargement which occurs in the course of certain forms of Bright's disease of the kidneys. As, however, nephritis acts also in other ways, the connection between disease of the heart and that of the kidney may be reserved for special consideration. Arteriosclerosis is a degenerative change in which the cellular elements of the capillaries and finer vessels are partially or completely destroyed, the protoplasm suffering hyaline, fatty, and atheromatous change. The blood-vessels assist the heart in the circulation of the blood; in fact, the blood-vessels are alone capable of circulating the blood. The recoil of the arteries upon the column of blood propels it mainly in the direction of the circulation on account of the closure of the valves at the orifice of the main trunks. This is a force which is felt only in the larger vessels. Degenerative changes in the smaller vessels interfere with the process of osmosis and thus directly increase the blood pressure. This increase of blood pressure under the general arteriosclerosis of age accounts for the enlargement of the heart which is so common in old people. The hypertrophy of the heart in old age is compensatory in the same sense as in valvular lesions. The increase in the size of the ventricles enables the heart to overcome the obstacles in the course of the smaller vessels. This increase in the size of the heart in the aged is possible only under a favorable nutrition, but as Leichtenstern has shown that hæmoglobin is always increased after the age of sixty, it is evident that age in itself furnishes certain factors which favor hypertrophy of the heart.

Charcot commented upon the fact that while every other organ in the body shrinks in volume in advancing years, the heart preserves its natural size, and in some cases undergoes a real hypertrophy. Cohnheim also noticed the fact that the heart of old persons does not,

as a rule, participate in the general atrophy of the body and especially of the muscles, but rather increases in mass and volume. The writer would subscribe to the view of Benneke, who believed that those individuals had the best chance to reach advanced life who are naturally endowed with strong hearts, and that age is not a possible inheritance of all, but only a select few destined to it from birth, notwithstanding the fact that Balfour cites this view only to refute it with the declaration that he has seen too many weak hearts and even hearts mechanically defective attain advanced age to be able to regard the idea as even approximately true. For arteriosclerosis is the most pronounced factor in the process of senile involution. The so-called natural death occurs by some process of arteriosclerosis. Anything which favors the development of arteriosclerosis precipitates the changes of age. These causes are chiefly hard work, alcoholism, syphilis, Bright's disease, diabetes, gout, saturnism, and psychical influences. The changes of arteriosclerosis are caused by the action of toxins carried to the blood-vessels through the vasa vasorum. These changes may be produced at once, as in the course of the acute infections, or may be induced gradually in the course of a chronic infection like syphilis, or they may creep on very insidiously as the result of defective nutrition from any cause. The evils of arteriosclerosis are longest counteracted by a strong action of the heart. It must be, therefore, that, other things being equal, the strong heart makes the individual fittest to survive.

(*c*) *Hypertrophy in Consequence of Muscular Effort (Hard Work).*— As already stated the heart corresponds, not only in size but also in force, to the demands of the tissues. The study of comparative anatomy shows that the size of the heart increases in proportion to the muscular effort. Bollinger made a number of these investigations, showing that animals which have to furnish enormous muscular power, and certain birds distinguished by velocity and long duration of flight, are endowed with larger development of the heart muscle. The smallest heart among birds is found in the heathpout, 4.09 per thousand. This animal lives a quiet and comfortable life on the earth. The buzzard, a lazy bird of prey, which only hunts mice, etc., shows 8.02 to 8.30 per thousand heart weight. The goose shows 8, the partridge 9.17, and the weight rises through the scale, according to the velocity and duration of flight, up to the thrush, whose heart weighs 25.6 per thousand of body weight. The horse, dog, chamois, hare, and deer show relatively heavy hearts. That of the deer, for instance, is twice as great as that of man. Müller proved that hypertrophy chiefly concerns the muscle of the left ventricle in draught, running, and hunting dogs in consequence of exces-

sive bodily strain. The same increase in volume occurs in man in correspondence with the character of the work. Thus Bauer called attention to the hypertrophy of the heart in the woodcutters of the mountains, the mountain climbers, hunters, etc. In climbing mountains a man becomes breathless because of momentary insufficiency of the heart, but a short rest suffices to restore the heart to perfect capacity. The difference in individuals in this regard, the ability to make sustained or powerful effort depends to a large extent upon the condition of the heart muscle. For a time the heart draws more upon its reserve force. This supply suffices for extraordinary demands made for a short time. When this supply no longer suffices, the muscular fibres multiply in number and size to meet the demands, and the heart compensates by its hypertrophy for the obstacle imposed upon the circulation by the general contraction of the muscles.

Germain Sée speaks of the hypertrophy of the heart in workingmen as the *cœur du travail*, which he observed in vocations requiring a sustained effort, as in blacksmiths, porters, musicians who play wind instruments, soldiers under forced marches, etc. Excessive or sustained contraction strains the heart and interrupts the circulation of the blood, but this evil is counteracted by the dilatation of the vessels which takes place in exercise. Oliver's studies with the arteriometer illustrate the feeding of the brain and body by this process. But the gymnast, the ball-player, and the bicycle-rider furnish a contingent of cases of excessive muscular contraction, and often of heart-strain, hypertrophy, and exhaustion.

In any case the demand may be at some time excessive. The individual will then suffer from an acute over-strain of the heart, which shows itself in the signs of insufficiency. Sometimes the heart is paralyzed outright. The individual may succumb to heart failure. This accident is especially liable to supervene when extraordinary demands are made upon a heart weakened from any cause. In other cases the individual may recover but incompletely and be left with a weakened heart. Such a heart is sometimes said to be "irritable."

Fraentzel records the case of a powerful hod carrier who was distinguished above his fellows by his herculean strength. They could carry regularly but thirty-six stones, each one weighing seven and a half pounds, while he could carry six stones more. Thus while they carried two hundred and seventy, he carried three hundred and fifteen pounds. One day he loaded himself up with six stones more than usual, and on lifting this weight he was suddenly seized with a pain in the left side of the chest radiating to the arm, so intense as to compel him to drop his hod and stop work. Hereupon he was affected with dyspnœa and palpitation to such degree as to

render it difficult for him to reach his home. This distress he suffered for several weeks, finding no rest day or night, when he was brought to the hospital affected with dropsy. He recovered in the course of sixteen weeks and returned to his work and gradually in the course of several months was restored to his original health and strength. Then upon some subsequent occasion he again loaded himself up with more than the usual amount, when, as he lifted his load, he was suddenly seized again with the same pain. He fell to the ground and could be carried to his house only in the course of an hour. Here he suffered the most intense dyspnœa and became affected with a general anasarca. He came to the hospital on the next day affected with cough and the feeling of great distress in the chest. On entry the dyspnœa was so pronounced that he could not lie down. The face and extremities were deeply cyanosed, the forehead was covered with a cold sweat. Cough was frequent and short with the expectoration of a thin, foamy fluid, lightly tinged with red. The slightest motion of the patient brought on symptoms of suffocation. The heart was found extremely enlarged and in a state of dilatation; the apex stroke was no longer visible but could be felt at times in the fifth intercostal space. The first sound was indistinct; there was exquisite delirium cordis, the pulse was correspondingly arhythmic. The face, even the tongue, was cyanotic. The urine was extremely scanty, brown-red in color, showed a brick-dust sediment, but no morphotic constituents. The patient again made a good recovery in the course of a long time, left the hospital apparently healthy, but was no longer able to do hard work.

The writer once experienced the effects of acute over-strain of the heart in the use of the forceps in a case of extremely difficult labor. The head had become wedged into the pelvis and was dislodged only under a supreme effort at traction, which was immediately followed by the signs of heart failure in exhaustion and faintness. For nearly a month any unusual effort with the arms or even the act of climbing stairs was attended with a feeling of weakness and distress in the region of the heart, palpitation, and a light degree of dyspnœa.

Peacock finds the heart dilated in many of the miners of Cornwall, who after the heavy hammer work of the day have to climb ladders of immense height in order to get out of the pit every evening.

These are the cases of acute heart strain where even the reserve force of the heart is rapidly exhausted, and dilatation ensues all the more rapidly because there has not been time for the development of hypertrophy. The character of the nutrition makes also a good deal of difference in the development of these cases. Where the food is insufficient or for any reason is not appropriated by the tissues, the heart shares in the general inanition, and anything like compensatory hypertrophy develops but imperfectly or not at all. On the other hand, an excessive food supply may directly favor hypertrophy of the heart. Here now may be considered

(*d*) *Hypertrophy in Consequence of Plethora.*—Any abnormal increase in the quantity of blood necessarily throws more work upon the heart. The quantity of blood is directly increased after every meal, as but the smallest quantity, perhaps in the proportion of an ounce to the pound, passes directly out of the body by the alimentary canal. All the rest of the food and drink is incorporated into the blood and juices of the body. Where the various processes of life are active, the consumption corresponds to the ingestion and the weight of the body remains practically the same. But when the mode of life is more inactive or sluggish, the quantity of blood accumulates to constitute the condition of true plethora. The blood-corpuscles may increase in number from four million to seven million to the cubic millimetre, and the hæmoglobin may increase from thirteen per cent. to sixteen per cent. There is even greater increase in the fluid element. As age advances the tendency to inactivity of the body increases. At the same time the appetite, instead of being impaired, is improved. The tissues actually demand more food to supply the waste of age. Frequently food is taken in excess and accumulations in the blood are favored by a more prolonged rest, sometimes by sleep, after meals. The increase in the quantity of blood is indicated in the fulness of the vessels, in the flushing of the face, headache, in the tension of the pulse, increased impact of the heart, attacks of palpitation and dyspnœa. The enlargement of the heart which develops in these cases may be readily perceived by physical signs. When with the increase in the quantity of food, whether absolute or relative to the wants of the body, large quantities of liquid are likewise ingested, the condition of plethora is all the more quickly or fully developed.

Individuals who have worked hard and have probably led abstemious lives, with the acquisition of a fortune easily drift into habits of luxury. The food is richer and more abundant, and is ingested with libations of wine. These individuals begin to gain weight, become fat and florid, and soon find that every effort is attended with palpitation and dyspnœa. The distress on the part of the heart is announced at first by occasional attacks of palpitation, as after dining out or at some public banquet. The sleep is disturbed by nightmare. There is a sense of fulness about the head, and vertigo in stooping over, as to button the shoe or in any act of straining, as at stool. The individual is said to become puffy and paunchy, and by this time the hypertrophied heart is beginning to flag in its efforts to force the blood through the distant vessels. So frequently is this condition observed in the upper classes as to be distinguished as the plethora of luxury, or what the German writers

call the "luxus consumption." The heir of luxury soon becomes familiar with the fact that life has its compensations in every direction, and the rich merchant, public official with a sinecure, and successful professional man learn that dignity may be secured without ease, and that *non in otio quies.*

Still it is a question if plethora alone may produce hypertrophy of the heart. Balfour scouts the idea, and declares outright there is in fact no physiology that teaches us that excessive nutrition promotes cardiac hypertrophy. There is no hypertrophy of the heart among the Strassburg geese, stuffed to repletion to supply the market with *foie gras.* Nor did any one ever hear, he says, of a young porker fattened for the butcher having enlargement of the heart. There must be some peripheral obstruction to start the hypertrophy. Sommerbrodt will not even admit the hypertrophy of work. He says there must be more to account for it, and appeals to the loss of the depressor reflex that makes the heart lose control of its capillaries.

Alcohol in any form acts in two ways in the development of hypertrophy of the heart. In the first place, alcohol is a frequent cause of arteriosclerosis, which in itself affords obstacle to the circulation of the blood. In the second place, alcohol acts as a chemical poison in the production of arteriosclerosis chiefly in the stronger forms, as in brandy, whiskey, etc., and increases the quantity of blood in a mechanical way chiefly in the weaker forms, as in wine and more especially in beer. Bollinger called especial attention to the great frequency of heart disease in Munich, with the statement that heart disease ranks in that city third as the cause of death. Sendtner had already observed that the mortality from heart disease of brewers and workers with beer was much greater than the general mortality. The blood pressure and pulse frequency are directly increased after the ingestion of any kind of fluid, but they are highest after beer, probably on account of its carbonic acid gas and alcohol, and possibly on account of the preponderance of potash salts. Next in descending series after beer are brandy, wine, coffee, tea, cocoa, water. Bollinger observed that after the ingestion of a pint of water during work by a strong girl aged twenty-two the blood pressure returned to the normal in the course of one hour, and after the ingestion of a half-pint of wine and a half-pint of water or of one pint of beer, under the same work, the blood pressure reached the normal only after two hours. The bloated beer drinkers of our own country nearly all suffer in the course of a few years from dilatation of the heart, the consequence of a previous hypertrophy. Billings showed that the mortality of dealers in liquor from twenty-five to sixty years of age from diseases of the circulatory system was 140, while that of men gener-

ally was 120 to the thousand. When the luxurious habit, more especially of the ingestion of alcohol, is associated with disease, like syphilis, which favors the development of arteriosclerosis, or when the heart is kept in a state of excitation by nerve irritants, strong coffee, tea, and tobacco, the effect upon the heart is even more rapid and certain. From his observations—and no one was ever more favorably situated to make them—Bollinger was led to believe that beer more than any other alcoholic drink develops the so-called idiopathic hypertrophy of the heart.

(*e*) *Hypertrophy in Consequence of Pregnancy.*—As already remarked, the claim made by Larcher and supported by a number of French observers, that the heart undergoes hypertrophy in pregnancy, was doubted by Gerhardt and seemed to have been overthrown by Löhlein, who weighed the hearts of nine women, of varying size, large, medium, and small, in puerperium and found the average to be 245 grammes. This weight corresponded exactly with that fixed by Clendenning in healthy women of average weight from the twentieth to the sixtieth year of life. On the other hand, Ducrest took the trouble to measure the heart in 100 women aged twenty to thirty, who had died in puerperium, and found that the wall of the ventricle was distinctly and decidedly thickened. Thus Bizot had fixed the average thickness of the heart in women at 0.010 mm. Ducrest found it in puerperium varying from a minimum of 0.011 to a maximum of 0.018 mm. The fact that hypertrophy does occur was re-established by Bollinger with an accurate study of 76 cases (1879–1890), in 67 of which the weight of the heart was established post mortem. Of these 67 cases, 9 died during pregnancy, 7 on the day of labor, 32 in the first week, 14 in the second, 1 in the third, 3 in the fourth, and 1 in the fifth week. Accurate measurement showed that the heart increases in weight in pregnancy and puerperium. The hypertrophy is most marked in young life, least in emaciated women. The increase in weight during pregnancy (8.8 per cent. of the normal weight) corresponds to the increase in weight of the body of the mother including the fœtus. Dreysel showed that the increase affected both ventricles and concerned the thickness of the walls as well as the diameter of the cavities. This increase continues uninterruptedly from the first months of pregnancy up to the day of labor. Then follows a reduction, rapidly at first, more slowly later. The cause of the hypertrophy is to be attributed to the increased work thrown upon the heart in supplying the mammæ, the uterus, and the placental circulation. Moreover, pregnancy increases the quantity of blood. This fact was demonstrated by Spiegelberg and Gescheidler in the case of pregnant dogs, and by Heissler in the case

of pregnant sheep, in which animal the increase is nearly one-fourth of the physiological quantity of blood.

(*f*) *Hypertrophy in Consequence of Disease of the Kidneys.*—Hypertrophy of the left ventricle belongs to Bright's disease as much as do dropsy, albuminuria, and uræmia. It is one of the cardinal symptoms of the disease, but it rises above the other signs—rises, in fact, to physiological dignity in that it expresses an effort to overcome or neutralize the main effects of the disease. A hard, wiry pulse, in the absence of fever or of any superficial evident disease, is enough of itself to excite this suspicion in the mind of the alert practitioner, and the sign is all the more valuable in that it shows itself early in the insidious course of this disease, months and sometimes years before nervous symptoms, dropsy, or failure of nutrition suggest the nature of the affection. For the increase in the impact of the heart is frequently to be observed in the course of two to four weeks from the inception of the disease, when this period may be determined, at which time it is already demonstrable on autopsy. It develops quickest and most completely in the healthy and robust.

Regarding the cause of the condition, Bright adopted the conservative view that it was caused either by the irregular and unusual irritation of the heart produced by the changed condition of the blood, or by the call upon the heart for increased activity to force the blood through distant contracted blood-vessels. Bright thus announced the two theories, chemical and mechanical, which still find advocates in the present day. In 1856 Traube published his support of the mechanical view in a paper that has been widely quoted ever since. He noticed the hypertrophy in connection only with the contracted kidney, and made it diagnostic of this form of Bright's disease. He derived it from the diminution in the quantity of blood flowing through the kidney, diminution also in the quantity of urine excreted, with the consequent accumulation of fluid in the aortic system and increase of blood pressure. Traube established the theory of compensation.

In refutation of this view, it has been shown, first, that after extirpation of an entire kidney or the amputation of an entire member, with the consequent ablation of a much greater number of capillaries, whole systems of capillaries in fact, hypertrophy of the heart does not ensue, as the blood finds escape to other vessels, in the case of the kidney to the other kidney, which enlarges to do double duty; second, that hypertrophy develops also in cases where there is no atrophy of the kidney substance, as in chronic parenchymatous nephritis; third, that there is no diminution, but, on the contrary,

an increase in the excretion of urine in the granulated kidney; fourth, that ligation of the ureters alone is followed by this hypertrophy.

But these objections have been more or less successfully met thus: First, that the cases of extirpation of the kidney have not been watched long enough to observe gradual and remote effects upon the heart; Grawitz and Israel in their experiments upon rabbits, and Lewinsky in his experiments upon dogs, in securing not sudden but gradual occlusion of the renal arteries, such as occurs in disease, invariably saw degeneration of the kidney structure ensue with hypertrophy of the left ventricle of the heart; second, that while this hypertrophy does follow parenchymatous nephritis, there is none the less occlusion in this form of Bright's disease; third, that while the quantity of water is increased in renal cirrhosis, the blood pressure is undoubtedly raised in the aorta to produce the increase in the water excreted; and fourth, that ligation of the ureters produces disease of the kidneys leading at times to obstruction of the circulation, and when this obstruction does not occur, as in cases of pyelitis, no hypertrophy of the heart ensues.

From this summary of the evidence, it must be admitted that the bulk of testimony favors the mechanical theory and puts this hypertrophy in the same position as that which ensues upon valvular defects, or as that which occurs in the right ventricle in consequence of any block in the lungs. It is difficult to subscribe to the chemical theory of hypertrophy, as advocated recently by Frerichs and Senator, in the face of the fact that uræmia or retention of any toxic elements of the urine from other cause, as from cystitis, pyelitis, etc., is not attended by hypertrophy of the heart.

But notwithstanding the immense amount of labor and research which has been spent upon this subject, the question is by no means definitely settled yet. Traube, as stated, attributed the increase in blood pressure to the destruction of extensive domains in the capillary system in the kidneys, together with an imperfect elimination of the water, but Traube himself was not willing entirely to exclude the influence of toxic agents. Johnson referred the hypertrophy to great thickening of the muscular tissue of the small arteries in consequence of tonic spasm of the vessels, and Gull and Sutton found the cause of the obstacle in a process of general arterio-capillary fibrosis with atrophy of the muscular tissue. The sclerosis in the kidney was considered as part of a general process. De Bove and Letulle attribute the hypertrophy of the heart in Bright's disease to minute scleroses of the myocardium, local lesions which are part of the general arterio-capillary fibrosis of Gull and Sutton, whose prin-

cipal seat is in the kidney. The sclerosis which is primitive entails the hypertrophy secondarily.

On the other hand, Bamberger called attention to the fact that the hypertrophy of the heart sets in early, before there is time for the destruction of the capillaries. So, Buhl reverses the relation, attributing the increase in the blood pressure to hypertrophy of the heart which develops first, partly in consequence of the disease in the kidneys and partly in consequence of a relative stenosis of the aorta. Ewald is not willing to accept this theory, because of the clinical fact that the increase of the blood pressure precedes the hypertrophy of the heart. Thus just when the mechanical theory seems to be established there is a resurrection of the chemical theory first suggested by Bright, attributing the hypertrophy of the heart largely to the irritation of toxic agents in the blood. The presence of these toxic agents has never been denied, though their exact character has never been established. Feltz and Ritter, from their examination of the blood of uræmic patients, attributed the chief effects to increase in the salts of potash. The injection of these salts, they claim, produces the symptoms of uræmia. These authors propose, indeed, to substitute for uræmia the name potassæmia. The poisoning by the potash salts they consider the cause of the hypertrophy.

A new and different solution of the difficulty has now been proposed by Israel. In this consideration it is remembered that the kidney is no mere filter, but is a secretory organ, and the suggestion is that the destruction of a large amount of secreting substance calls for extra work upon the heart to supply a larger quantity of blood to the diminished areas.

Thus while the existence of hypertrophy of the heart in chronic nephritis is so universally acknowledged that the condition of the heart is accepted as a sign of the disease of the kidneys, and the polyuria of certain forms is directly attributed to the force of the heart, the *modus operandi* of the process is by no means plain. More light yet is needed to disclose additional facts which will reconcile discordant views and clear the subject up.

Diabetes produces enlargement of the heart through arteriosclerosis, as this particular degeneration of the vessels is not infrequently due to this cause. Israel found hypertrophy of the left ventricle in ten per cent. of the cases of diabetes, Mayer and Saundby in thirteen per cent. The hypertrophy of the heart was found also in connection with disease of the kidney. Meyer found hypertrophy of the whole heart in 24 of 90 cases of diabetes (twenty-seven per cent.) during life (von Noorden). Frerichs has shown that heart failure entirely independent of coma is a not infrequent cause of death in diabetes.

The final fatal failure follows immediately upon some unusual exertion or emotion.

Gout almost universally affects the heart. The older writers speak of gout of the heart. The heart is affected generally in connection with the kidneys. The hypertrophy of the heart is usually considered a sequel of the cirrhosis of the kidney. The modern view is rather to the effect that the same cause produces both conditions. At least the pathological process in both organs is an expression of interstitial sclerosis. Atheroma of the coronary arteries is very frequent in gout. All the conditions are favored by the fact that they occur in advanced age, which is in itself an important element in the production of arteriosclerosis. An irregular and tumultuous action, with precordial pain sometimes so intense as to be regarded as an angina, occur frequently in the history of gout. Sooner or later the hypertrophy gives way and dilatation ensues. The patient suffers with catarrhal affection of the lungs, with frequently recurring attacks of dyspnœa, with cardiac asthma, stasis in the liver and kidneys, dropsy, and progressive marasmus. The muscle fibres suffer degeneration, fat accumulates on the surface and insinuates itself between the fibres; the muscular fibres themselves undergo granular and fatty degeneration, while the vessels show atheromatous and sclerotic change.

(*g*) *Hypertrophies in Consequence of Diseases and Deformities of the Chest.*—The hypertrophied heart which occurs in consequence of disease of the lungs concerns especially the right ventricle and is observed first of all in connection with arteriosclerosis, a process which has been sufficiently described. The most frequent congenital anomaly of the heart is, as stated elsewhere, stenosis of the pulmonary artery, at its origin or in its course, and this stenosis necessarily leads to hypertrophy of the right ventricle, which becomes sometimes so great as to overshadow the left, even in extra-uterine life.

Germain Sée called attention to the hypertrophy of growth, which he described as a precocious development of the heart under a tardy development of the thorax. This disproportion disappears with age, but if it should persist, it entails interference with the pulmonary circulation, and hypertrophy of the right heart. Bloch set out to establish the reality of the hypertrophy of growth, but retreated and took refuge in hereditary predisposition. This predisposition shows itself in children of tuberculous, alcoholic, and neuropathic parentage. Such children show degenerated hearts, and hypertrophy may set in, in these cases, as early as the age of seventeen years.

Chronic catarrhal processes, emphysema, asthma, chronic bronchitis, especially when of frequent recurrence, affect the lungs in the

same way and lead to the same results. So also acts compression from without, as by pleuritic effusions. In the diseases of most frequent occurrence, pneumonia and tuberculosis, the heart is differently affected in different cases. In croupous pneumonia the main force of the poisons is expended upon the heart, but the course of the disease is too quick for the development of hypertrophy. The pneumonia which results from the influenza bacillus or from pyogenic micro-organisms runs a more chronic course and is more liable to be followed by hypertrophy of the heart. Those cases especially which terminate in induration of the lungs are marked by hypertrophy of the right ventricle.

In tuberculosis the heart is sometimes too large and sometimes too small. From his studies Oppenheimer finds the small heart at puberty both absolute and relative, *i.e.*, a congenital condition and not a consequence of emaciation, as the main predisposing cause to tuberculosis. Reuter found in 179 male patients 72 (40.2 per cent.) with hearts too large; 55 (30 per cent.) with hearts of normal size; 52 (29 per cent.) with hearts too small. So that while small hearts may favor the development of tuberculosis, large hearts do not prevent it. It is in the chronic and insidious cases of tuberculosis, of very slow development, marked by long periods of quiescence, during which the lung tissue shrivels and the diseased portions are affected with the so-called fibroid degeneration, that hypertrophy of the heart is chiefly developed.

But of all the diseases of the lungs none is so well calculated to induce hypertrophy as emphysema. In this disease large tracts of capillaries are obliterated by the compression of the distended air cells, are often in fact congenitally defective, so that great work is thrown upon the heart to supply the lungs with blood enough for aëration of the tissues. The influence is exaggerated further by the fact that cases of emphysema are liable to attacks of acute bronchitis and often to explosions of asthma, which superadd the element of muscular spasm and contraction. So that the most exquisite cases of hypertrophy of the right ventricle are found in connection with emphysema of the lungs. Unfortunately, the condition of the heart may not always be established by physical signs, because the increase in the size of the heart is concealed by the overlying distended lung.

The frequency of bronchial affection in childhood led Bollinger to believe that children who die of any disease are liable to show moderate hypertrophy and dilatation of the heart. This author cites from Steffen the report of 40 cases of hypertrophy and dilatation of the heart in children. In these cases the left heart was found alone affected eight times, the right twice, both together thirty times.

Some affection of the lungs was found in all cases of hypertrophy of the right ventricle. Sometimes there are evidences of pleurisy, pneumonia, tuberculosis, or emphysema; in certain cases there was evidence of pre-existing swelling of the peri-bronchial and mediastinal glands, which had since subsided, but which had acted mechanically upon the pulmonary artery and its branches to produce stasis in the right heart.

Mechanical deformities in the same way throw extra work upon the heart. All cases of pronounced deformities of the vertebral column are attended with hypertrophy of the right ventricle. In the observations of Neidert of 31 cases of kyphosis and kypho-scoliosis, the heart was found perfectly normal only six times, and in all these cases the deformity was but slight. Of the 31 cases, 8 died of tuberculosis, 4 of myocarditis, and 7 of acute heart failure. The conclusion of Neidert that marked deformities of the vertebral column proved fatal by exhaustion of the heart, was supported by the studies of May, who made frozen sections to show the situation of the viscera in scoliosis.

Draught animals always have large hearts with an especial development of the right ventricle. These facts are in accord with certain observations from comparative anatomy. The relative weight of the heart in the thrush, four to five times greater than that of man, is attributed to the great strain upon the lungs in singing; and the breadth and development of the right ventricle in the dog probably stands in connection with the function of the lungs, as in consequence of defective secretion of sweat in the dog the lungs must act as regulatory organs for the body temperature. Besides there is strain on the part of the lungs, that is of the lesser circulation, in the dog in hunting, in long-continued barking, and in accompanying equipages, etc. (Bollinger).

III. Hypertrophy due to Disease of the Heart Muscle from Degeneration, Infection, Neoplasms, etc.

(*a*) *Hypertrophy from Fatty Degeneration.*—The various degenerations which the heart undergoes in consequence of acute and chronic affections are nearly always attended with increase in the volume of the heart. In the process of fatty degeneration the muscular tissue itself suffers retrograde change, whereby the active protoplasm is substituted by inert fatty matter, and by fat which is usually insinuated between the muscle fibres. During the process of degeneration the heart muscle undergoes a compensatory hypertrophy in unaffected regions. The incompetence of the muscle structure under

molecular substitution by fat is indicated in the dilatation which the heart rapidly undergoes and which may be so great as to lead to relative insufficiency of the valves. This will be referred to again in the section on Fatty Heart.

(*b*) *Hypertrophy from Myocarditis.*—Hypertrophy occurs in the same way in the various degenerations of myocarditis, and enlargement of the heart is usually seen in the course of diphtheria, scarlet and typhoid fevers, the most frequent causes of myocarditis. Jaeger found in diphtheria and scarlet fever dilatation and hypertrophy of the heart, in diphtheria especially of the right ventricle. Penzoldt speaks of the acute dilatations which occur in this group and which belong in "a characteristic and tragic way" to diphtheria. Jaeger found at the children's hospital at Munich (1882–1893), in 615 autopsies of diphtheria, gross evidence of nephritis in 28, *i.e.*, 4.55 per cent. of cases, and of these cases 13, *i.e.*, 46.4 per cent., showed hypertrophy of the heart; while in 47 autopsies of scarlatina nephritis was found 21 times, *i.e.*, 44.68 per cent, and of these cases 14, *i.e.*, 66.6 per cent., showed hypertrophy of the heart. In scarlatina hypertrophy is most pronounced, while in diphtheria dilatation prevails, because probably of the more profound toxic effect of the diphtheria poison upon the muscle of the heart. The most acute dilatations have been observed in diphtheria, typhus fever, pneumonia, erysipelas, and dysentery. As the heart muscle is rapidly weakened in these affections, the enlargement is chiefly the result of dilatation, but for a time the heart endeavors to counteract the weakness of its structure by increase in its volume. Under defective contraction the ventricle does not empty itself entirely. There is of necessity, as Roy and Adami call it, residual blood in the ventricular chambers. These acute observers found, in fact, in their experiments on the lower animals that "when they pushed the little finger into the cavity of the ventsicle it could be felt that while the walls of the ventricle in the lower two-thirds up to the apices of the papillary muscles close completely around the finger, there is a clear space in the upper third which is not and cannot be emptied of blood. These observers show, therefore, that hypertrophy is never primary, but is always preceded by dilatation. They thus give the death-blow to concentric hypertrophy. Hypertrophy is always, of necessity, eccentric. Krehl finds from his studies that inflamed muscles contract badly—a condition which might have been predicted. He calls attention to the fact that acute myocarditis reduces the working capacity of the heart and attributes the enlargement chiefly to proliferation of connective tissue. In all the nine hearts he subjected to critical examination there were found anatomical changes. The striated substance showed often

intense fatty degeneration and granulation, with reduced capacity to take up coloring matters. The pigment of the muscle fibres is much increased. In all the hearts the connective tissue was increased about the vessels. This connective tissue was often rich in cells and constituted a pure infiltration between entirely normal muscle fibres. Round-cell infiltration was especially marked in certain cases and some of the vessels showed a light degree of sclerosis, which is evidence "how near this condition is with that which produces arteriosclerosis." Krehl is inclined to ascribe to myocarditis—that is, to anatomical changes—in the heart tissue the chief rôle in the production of most of the cases of so-called idiopathic hypertrophy.

The development of dilatation in the process of chronic myocarditis admits of easier explanation, as it may readily be attributed to the substitution of the heart muscle fibre by connective tissue. Under this substitution the heart loses its power of contraction, becomes distensible and is readily dilated. This sclerotic degeneration is noticed more especially in the left ventricle, and the evil of it —that is, the incompetence of the left ventricle—is counteracted for a time by hypertrophy of the right ventricle. In the course of time the right ventricle itself gives way, and the signs of dilatation of this structure are indicated first by tachycardia with arhythmia and by attacks of palpitation and dyspnœa. Thus Penzoldt quotes from Ebstein the observation that a considerable hypertrophy of the heart muscle could always be demonstrated in cases in which the activity of the heart was sustained in regularity up to death.

In this connection may be studied also the hypertrophy which occurs in connection with syphilis. Syphilis may enlarge the heart by the direct deposit of gummatous tumors, but more frequently in the processes of arteriosclerosis. Thus syphilis is a factor of great importance, ranking next, in fact, to alcohol in the production of cicatricial myocarditis. This condition soon renders the heart incompetent, and sudden heart failures, marked by palpitation, dyspnœa, sometimes by cyanosis, oftener by precordial pain, may immediately precede heart failure and sudden death. In some of these cases death occurs actually suddenly without any previous intimation or sign of infection of the heart. More frequently the process is slower, and the first indication of infection is usually an early exhaustion. In these cases there is more or less dyspnœa upon exercise, and considerable œdema shows itself about the feet at night.

Syphilis of the heart belongs among the later manifestations of the disease. It may therefore show itself in the absence of other lesions. But the history of the infection can usually be traced under a proper examination. See the action on Syphilis of the Heart.

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The enlargement of the heart which is sometimes seen in connection with pericarditis is due wholly to associate myocarditis. Any extensive inflammation of the pericardium is always attended by inflammation of the underlying zone of the muscular tissue itself, with a consequent hypertrophy and dilatation. But as already stated, when the pericarditis is so extensive as to become adherent to the wall of the sternum or the mediastinum, the interference with the action of the heart may be so great as to lead to compensatory hypertrophy.

(*c*) *Hypertrophy from Tumors and Parasites.*—The tumors of the heart will be elsewhere briefly considered. As the name implies, a tumor from its very nature enlarges the heart. Thus myomata, cystic tumors, or malignant growths may produce distinct deformities, and in rare cases parasites, cysticerci or echinococci, may be deposited in the substance of the heart. Aneurysm occurs in the wall of the heart as well as in the course of the great vessels. Besides the mere mechanical enlargement produced by the presence of tumors, the heart undergoes hypertrophy in the endeavor to overcome obstacles which they offer in the course of the circulation. Thus extensive hypertrophy of the auricles and enormous hypertrophy of the ventricles has been observed in these conditions.

IV. Hypertrophies in Consequence of Diseases of the Nervous System.

The innervation of the heart is studied in detail in the secction on the Cardiac Neuroses, where emphasis is laid upon the fact that while the heart muscle contracts automatically the force and frequency of its action are regulated by the nervous system. Mere increase in frequency in the action of the heart does not necessarily produce hypertrophy or dilatation. Cases of tachycardia of months' duration do not, for instance, develop hypertrophy or dilatation of the heart. Hypertrophy is more wont to ensue when the stimulus is applied periodically, as the heart has, during intervals of rest, opportunity for recovery and restoration.

(*a*) *Hypertrophy from Mechanical Irritation of the Vagus.*—Where the vagus nerve is subjected to irritation, hypertrophy of the heart is wont to ensue, but any long-continued irritation would act like an excessive strain, exhaust the heart and lead to dilatation. Thus irritation at the origin or in the course of the trunk of the vagus, as from sclerotic changes in the medulla in the course of bulbar affections or irritations of the trunk of the nerve by the pressure of tumors, lymphatic glands, etc., may lead eventually to hypertrophy of the heart.

Whatever may be the theory of its origin, Basedow's disease is

the most frequent affection attended by enlargement of the heart. The change in the outlines is not apparent at first, but ensues after the development of the goitre and exophthalmus and the existence of palpitation for some time. It is noticed then that the heart is big; the dulness reaching sometimes to and beyond the right border of the sternum, while the apex is dislocated downward and to the left. The enlargement here, however, is not so much an hypertrophy as a dilatation, which soon supervenes, as is evidenced by the paroxysmal attacks of dyspnœa, and more especially of extreme frequency of the pulse, tachycardia, which is regarded as a sign of acute dilatation. Cases of incurable Basedow's disease succumb usually to heart failure.

But the vagus may be excited also in a reflex way. Thus Potain finds a number of cases in the literature where nerve injury, especially to branches of the left brachial plexus, was followed through reflex excitation of the vagus by attacks of angina pectoris with hypertrophy of the left ventricle. The reflex excitation first induces contraction of the peripheral vessels and thus leads to overdistention with compensatory hypertrophy of the left ventricle. Proper treatment of the neuralgia, extirpation of neuromata, resection of nerves, the administration of the bromides, etc., relieve the angina and resolve the hypertrophy. This arterial constriction may be produced further by stimulation of various parts of the nervous system, and especially by irritation of the abdominal organs. Hence chronic inflammation of the organs in the pelvis, protracted catarrh or constipation suffice, through the action of the abdominal sympathetic nerves, to induce contraction of the arteries and increase resistance in the circulation.

(*b*) *Hypertrophy from Chemical Irritation of the Vagus.*—Under this head may be considered the action of alcohol, which has already been studied in connection with the changes in the vascular system and increase of muscular effort, and of tobacco, coffee, tea, and stimulants of this class. Tobacco affects different people in different ways. Most people are poisoned by it at the start. Ordinarily tolerance is begotten in time and the individual may indulge in the use of tobacco, even to excess, without any symptoms of distress other than occasional nausea. Certain individuals may use tobacco to excess without any perceptible effect upon any organs of the body, but as a rule excess is punished in the long run. It is a common experience that an individual may use tobacco throughout the whole period of adolescence and maturity with perfect impunity, whereupon, at the age of forty to fifty, may ensue all at once some of the toxic effects of the drug. If he may pass this period without suffer-

ing, he may be able to smoke without fear the rest of his days. Certain of these effects which are characteristic are affections of the nervous system of the heart. Tobacco acts in some cases upon the motor and in others upon the sensory apparatus of the heart. Certain individuals are affected with neuralgia of the heart, sometimes so extreme as to simulate a genuine angina pectoris. In other cases only the motor apparatus is effected and the heart is thrown into palpitations or shows arhythmia or tachycardia. The tobacco neuroses of the heart occur more especially after smoking strong cigars in great number, often after the use of one cigar after another during the whole course of the day. Such smokers may be then seized all at once during the night with a neuralgia of the heart or with a severe attack of palpitation. Attacks after chewing tobacco are less frequent, but one such case occurred in the practice of the writer, and the attack, which was the culmination of a series of prodromes, showed itself with such severity when it occurred as to cause the patient to abandon his filthy habit at once and forever. It is said that, though other symptoms of tobacco poisoning may ensue, these attacks of neuralgia and palpitation of the heart never occur after the use of cigarettes.

Coffee is a direct stimulant of the heart. The active principle of it, caffeine, exercises its chief influence on the nerves of the heart. The use of strong coffee invariably brings on an attack of palpitation in some people, and the excessive use of coffee leads finally to exhaustion of the heart. Tea acts in the same way, though it is a milder beverage and is therefore oftener indulged to excess. Palpitation of the heart belongs among the common symptoms of the "tea-drinker's dyspepsia."

(*c*) *Hypertrophy from Psychic Irritation.*—The vagus nerve slows the heart and may reduce the output as much as thirty per cent. Such a reduction as that leaves blood in the heart. The nervous mechanism of the heart is adjusted with infinite nicety. The heart is made for the body. The capillaries are formed first in the tissues, then come the smaller vessels, next the larger vessels, finally the heart, of the proper size, weight, and capacity, in every way in proper proportion to the wants of the tissues. The tissues signify their wants to the heart.

The heart also has a way of signifying its capacities. It can, through the arterioles, control the calibre of the capillaries, but if these vessels are sclerotic they cannot respond to the heart, consequently so far from assisting in the circulation of the blood by their own resilience they throw extra work upon the heart by resistance. Further, stimulating and inhibitory influences go to the heart from

all parts of the nervous system. It is true that there is no tonic like pleasure. In the same way the heart feels the influence of depressing emotions. Balfour speaks of the pathetic manner in which life is every day shortened by the petty troubles, anxieties, and worries which are of daily occurrence and which by continual inhibition impair the ventricular systole and favor dilatation of the heart. "There are few of us," he says, "who have been in practice for even but a short time who have not had occasion to note the development of serious cardiac symptoms from the trouble arising out of untoward domestic affairs, the worry of an unsuccessful business, or even the wear and tear of a too successful business which has outgrown the physical powers of its manager." Leyden distinguishes two stages of this affection of the heart, which is produced *non vi, sed sæpe cadendo*, the stage of erethism and that of organic dilatation.

(*d*) *Hypertrophy in Consequence of Excess in Venery.*—Excess in venery exhausts the heart by both physical effort and nervous strain. Such excess is most frequently indulged in by young men, and attacks of palpitation and exhaustion, associated in the course of time, with hypertrophies of the heart, are occasionally observed. These conditions are sometimes seen in young married life, but are more frequent when unnatural stimulus is sought in illegal relations. Coitus prolongatus and coitus reservatus sooner or later affect the heart. The physician is consulted most frequently perhaps by older men, especially by older men with young wives, or by the habitual roué and débauché. Women are almost never affected in this way, though the condition has been reported in exceptional cases, especially among prostitutes and sexual perverts. Children or young people of either sex who indulge in masturbation to excess may suffer in the same way. The writer has seen much palpitation and precordial anxiety from this cause by reason of its depressing psychic effects.

Combined Causes.—As already intimated, two or more causes may combine to produce hypertrophy of the heart, or the same cause may act in different ways. Thus the effect of overstrain has been studied in connection with the raising of the blood pressure. Cases have been cited also of direct paralysis of the heart under excessive effort. Long-continued muscular effort, though less excessive, would produce the same effect. Reference has been made also to the hypertrophy of the heart which occurs in wine and beer drinkers; and while this hypertrophy has been attributed partly to chemical irritation it is due also, to some extent, to the heavy work of the drinker in his avocation and to the excessive work which the heart is forced to do under this stimulation.

Germain Sée speaks, as stated, of the hypertrophy of the heart in workingmen as the "*cœur du travail,*" which he observed in vocations requiring a sustained effort, as in blacksmiths, porters, musicians who play wind instruments, soldiers under forced marches, etc. But the work is not the only factor.

Hypertrophy from overstrain may thus result from the excessive use of the bicycle, especially by individuals who live sedentary lives during the week and indulge in this exercise to excess on holidays and Sundays. Muscle work and abundance of fluids together greatly increase the blood pressure (Maximowitsch, Rieder).

Factors favoring the increase of blood pressure in the vessels are the continuous strain of respiration during limitation of the respiratory movements, for instance by heavy knapsacks, sword belts, fatiguing marches, prevailing cold, as in the campaigns of '70 and '71, which leads to contraction of the peripheric vessels, as well as the unavoidable psychical excitement (Penzoldt). The veteran stands the exercise perfectly well, but the recruit is often greatly exhausted after the morning drill. This difference is to be attributed to the development of the heart under regular exercise. But as certain soldiers suffer sooner or more than others, Herz holds that these sufferers are originally endowed with weak hearts. The fact is, to make the heart hypertrophic by severe bodily effort, there must be peculiar circumstances which influence the organ, one might say, injure it. The tendency is to think with Sommerbrodt, that in some way or other the reflexes which can reduce the action of the heart have fallen out. Such reflexes are certainly discharged from the heart through the depressor nerve, but they may have their origin in other parts of the body, for instance in the striated muscle itself (Krehl).

Symptoms.

Hypertrophy of the heart reveals itself by increase in the size of the heart and by the effects of increase in the force of its work. The increase in the size of the heart manifests itself by dulness which extends to the right of the sternum and to the left of the mammary lines. In a pronounced case the increased dulness is readily recognized, but in slight degree it is diagnosticated only with great difficulty. Anything like an appreciable enlargement of the heart shows itself first in deviation of the apex stroke. Usually the apex stroke is said to fall two inches below the nipple (in the male) and one-half an inch to the right of the left mammillary line. Unfortunately, the position of the nipple is not as fixed as the umbilicus, and this structure or point is not so valuable in determining anatomical data.

Then it must be remembered that the heart itself changes its place. As the heart swings in the cavity of the thorax suspended only by the great vessels which issue from the base, the apex itself moves from right to left half an inch to an inch with change of posture from one side to the other.

Hypertrophy of the left ventricle reveals itself first by slight deviation of the apex to the left. Obesity, or, later, anasarca or hydrops, may conceal or obscure the increase in size, but nothing can conceal or obscure the dislocation of the apex beat. A heart which shows its apical stroke below or to the left of the nipple is either enlarged or out of place. Various extrinsic causes may dislocate the heart, but they may be more or less eliminated in the history of the case. At least a pleuritic effusion or tuberculous change in the lung tissue, the most common extrinsic causes, should not long embarrass a diagnosis. Hypertrophy from endocarditis would distinguish itself by a characteristic bruit as well as by accentuation of unaffected valves. A dislocation of the apex in consequence of the elongation of the aorta, which occurs in aneurysm, would be differentiated by the signs characteristic of aneurysm.

The same increase in volume which dislocates the apex also increases the force of the heart, so that the impact is felt over a greater area. A slight degree of hypertrophy may pass unappreciated in this regard, especially in the presence of much muscular development or in connection with a good panniculus adiposus. In pronounced degree the hypertrophied heart visibly agitates the whole precordial region, and in extreme cases, such as are observed usually only in connection with valve lesions, the force of the heart is so great as visibly to lift the head of the auscultator. Perhaps it is not unfair to say that moderate hypertrophy of the heart makes itself manifest to most observers by the increased area, not of dulness, but of impact to the hand laid flat upon the surface of the chest, or to the face in auscultation of cardiac sounds.

Percussion does not furnish such definite information as is claimed; for an hypertrophy which would increase the thickness of the ventricle as much as one-half a centimetre would be very decided, but would show no increase in the area of dulness. Percussion is not fine enough to appreciate such subtle change. As soon as dulness is so extensive as to be distinctly appreciated, either to the right or to left, it is a case rather of dilatation than of hypertrophy. So it may be said that hypertrophy makes itself manifest sooner by deviation of the apex than by increase in percussion dulness.

The next most valuable sign of hypertrophy of the heart is accentuation of the second aortic tone. It was Traube who first of all

recognized the fact that the blood pressure was raised in these cases, either as the cause of the hypertrophy of the heart or as the common effect of some obscure process. But from whatever cause, the effect must be the same and the reason of the accentuation of the aortic tone becomes obvious. The hypertrophied ventricle forces its blood into the aorta, which is already distended, and the closure of the aortic valves follows under pressure with a quick, short click, in fact, with a distinct accentuation, audible all over the heart, but in greatest intensity at the base, on the right of the sternum at its junction with the second rib. Now, just as accentuation of the pulmonic valve sound, which is heard in its maximum intensity on the left of the sternum, points to hypertrophy of the right ventricle, so does accentuation of the aortic sound point to hypertrophy of the left ventricle.

The increase in the force of the heart shows itself also in its effects upon the general circulation. As already stated, the blood pressure is increased. The radial pulse is then full and hard and in many cases hypertrophy of the heart is first suspected, or the diagnosis is actually established by the hard, incompressible pulse at the radial artery.

These four signs—increased tension of the radial artery, dislocation of the apex to the left, increased impact (and dulness when it may be appreciated), and accentuation of the second aortic tone—constitute the cardinal signs of hypertrophy of the heart.

But the compensation of the hypertrophy is not always exact. It may reach extremes in both directions, *i.e.*, of excess and of failure. In otherwise healthy, vigorous subjects, more especially in subjects leading an indolent or sedentary life, or in children with abundant nutrition, the hypertrophy reaches a degree at times superior to the demand. Attacks of violent palpitation may then ensue with even audible throbbing and marked increase of impact which makes itself manifest in yielding chest-walls as visible elevations or prominent bulgings of the surface. Hyperæmia of the brain occurs in these cases, with intense headaches and vertigo entirely independent of uræmia, and epistaxis may be frequent and severe. Strümpell reports two cases of fatal nose bleed from this cause.

On the other hand, in constitutions enfeebled by original construction or by acquired disease, the cardiac hypertrophy may fail to develop, and in all cases sooner or later it must fall short under the gradually increasing demands. The nice compensation which has hitherto sufficed to obviate the main danger of nephritis, the retention or non-secretion of sufficient urine, becomes disturbed, and the signs of accumulation of toxic elements speedily ensue.

The first sign of a flagging heart is noticed in the pulse, which

loses its hardness to become frequent and small. The great evil of stasis in the lungs from incomplete emptying of the left ventricle, accumulation in the left auricle and pulmonary veins, is counteracted for a short time by hypertrophy of the right ventricle which now sets in. But it is a work of Sisyphus that is thus thrown upon the right ventricle. It must at some time yield to the strain, and dilatation shows itself in the attacks of bronchitis, dyspnœa, asthma, and œdema pulmonum which soon supervene. There is headache from anæmia of the brain, and general dropsy, which commences about the ankles and eyelids, mounts to the genital organs, and invading the serous sacs, finally puts an end to the scene.

In all cases the main point is the recognition, not of hypertrophy, but of dilatation. Dilatation is the first sign of heart failure. When the heart fails everything fails, and the beginning of dilatation is indicated by the early occurrence of exhaustion and fatigue in any kind of effort. Perhaps attention is first called to the fact by the panting respiration which occurs after some unwonted effort, as in running to catch a car or train, or the individual notices that he is short of breath in climbing stairs. An attack of palpitation of the heart sets in after a full meal, or after the use of alcohol, tobacco, or some of the agents which act upon the heart in a toxic way. Hereupon gradually ensue the coarser signs of heart failure just described.

Sometimes the dilatation is more acute, especially under the influence of the nervous system. Jacob recently described the train of symptoms which marks this form. The attack begins with pains in the limbs or abdomen, anxiety, followed by pain in the region of the heart, dyspnœa, sometimes vertigo, cloudy vision, in extreme cases with profound unconsciousness, or the feeling that the heart is standing still or is beating more frequently or severely. The skin is cool or cold. During the vertigo the pupils are dilated, the pulse is small, the heart is either retarded or markedly increased, the breathing is rapid. The heart is found dilated and sometimes there are signs of œdema of the lungs. The attack lasts for hours or days and ceases with returning warmth to the skin and sweat. The pulse becomes soft and full, and the heart returns to its natural frequency in the course of a few days. Œdema and albuminuria disappear. This acute dilatation is caused by spasm of the vessels in the domain of the aorta, which produces all the symptoms, including dilatation of the heart, and the condition is appropriately designated an acute angio-spastic dilatation of the heart.

Allbutt relates from his own experience how on one occasion, toward the end of a long day's mountaineering in Switzerland, he was rather suddenly seized with a strange and peculiar *besoin de res-*

pirer accompanied by a very distressing sense of distention and pulsation in the epigastrium. On placing his hand over the heart, he felt a laboring, diffused beat all over the epigastrium, and by percussion he ascertained that the right ventricle was very greatly dilated. He threw himself on the grass with his shoulders raised, and in a few minutes had the satisfaction of finding the distention, the oppression, and the dulness recede. He could then run and even move about on the level, but the instant he began to ascend the symptoms returned, so that it was only with great caution that he could proceed. During the following night he was awakened again with severe palpitation and dyspnœa, which, however, passed off as soon as he went to the window and drew a few deep inspirations.

The evidence of dilatation is not so distinctly manifest in the physical signs. As already stated, the appreciation of any increase of dulness indicates the state of dilatation. The apex is dislocated but the force of the heart is diminished. In this point the observer may be easily misled as the heart may be stimulated to unwonted activity at the time of examination, and irregular and tempestuous actions, which are really signs of weakness, are sometimes mistaken for increase of force.

The truth is, increase of frequency is nearly always associated with lessening of force. The dilated heart always beats more rapidly and as stated elsewhere, cases of real tachycardia, in which the pulse is increased up to 200 and more, are nearly always due to dilatation of the heart. Increase in the frequency of the pulse is, therefore, an early, common, and characteristic sign of dilatation of the heart.

Not at all infrequently the dilatation becomes so extreme as to render the valves incompetent. Such a defect in the closure of the orifices, independent of diseases of the valves, is known as a relative incompetency. As mitral regurgitation is the most common affection of the valves of the heart, and as this defect is compensated for a considerable time by an hypertrophy of the right ventricle, upon which in the course of time dilatation must ensue, relative incompetency is observed first and most frequently in the tricuspid valves. It is, therefore, not at all uncommon to be able to hear a bruit at the ensiform cartilage indicative of relative incompetency of the tricuspid valves in the entire absence of any evidence or history of affection of these valves.

Diagnosis.

The diagnosis of hypertrophy is determined by the four factors mentioned: by the dislocation of the apex, by the accentuation of the aortic valve sound, by the increase in the outlines, and by the

condition of the radial pulse. To these signs may be added the visible effects of an increased blood supply to the various organs, especially the occurrence of headache, epistaxis, polyuria, etc.

Sometimes the genetic relationship of the originating malady throws light upon the subject. Thus hypertrophy of the heart is always present in chronic nephritis and in arteriosclerosis.

The essential factor in diagnosis is the recognition of the occurrence of dilatation. This stage is indicated, as stated, by the signs of weakness, by increased frequency of the pulse, by attacks of tachycardia, palpitation, a little later by dyspnœa upon exercise; by the evidence of stasis in the kidneys, that is, by the reduction in the quantity of the urine, which is high-colored, full of sediment and contains hyaline casts; and in the liver, which is usually distinctly enlarged; still later by the more severe dyspnœa and by the dropsies, which commence at the feet and extend up the trunk.

The most practical point in connection with hypertrophy and dilatation is the discovery of the cause. Attention is paid here to the history and habits of the individual; to the existence or pre-existence of syphilis; to the use of alcohol, including the ingestion of beer; to the existence of Bright's disease as determined by the examination of the urine; to excess in the use of tobacco, coffee, tea, etc., especially to the presence or absence of lesion of the valves. Digitalis exercises such a direct effect in the relief of the signs of dilatation in the absence of valve lesion that it may be taken as an assurance of the diagnosis. Under an increased diuresis improvement shows itself often in twenty-four hours with gradual disappearance of cyanosis, œdema, albuminuria, so that the incompetent and irregular heart becomes normal in its action at least.

Pericarditis shows dulness in a particular form, weakening of the impact and of the apex stroké, which may be sometimes unappreciable, with friction sound, fever, pain, disturbance of circulation, etc. A doubtful case may be determined by aspiration.

Schott suggests in cases of doubt the practice of gymnastics, which in a short time reduces the size of the heart and relieves all the symptoms of dilatation, but has no effect upon the pericardial exudation. Irregular exercise may hurt the heart, but a systematic and graded exercise always strengthens it.

Aneurysm of the aorta may be distinguished by dulness in the region of the aorta, by the thrill, bruit, etc. Aneurysm of the aorta may, however, be associated with hypertrophy of the heart.

Prognosis.

From whatever cause it may occur, hypertrophy of the heart is differently interpreted by different physiologists. Hypertrophy has long been regarded as a physiological process, as an overgrowth which overcomes obstacles. It is always spoken of as a compensatory process, and is regarded with favor by the clinician, who considers the outlook good always so long as the hypertrophy is marked, and makes his prognosis bad only when the hypertrophy begins to give way, when the heart muscle suffers degeneration and the heart chambers become dilated. Trousseau spoke of hypertrophy as "a lesion specially and providentially employed by nature as a means of overcoming the obstacle to the circulation of the blood," and Balfour mentions an acquaintance with a hale old gentleman, eighty-six years of age, who for sixty-six of these years was known to have suffered with a dilated and hypertrophied heart. Sixty-six years, he says, is certainly the longest period in his experience that mitral regurgitation with hypertrophy has been known or even surmised to exist.

But this view does not remain undisputed at the present time. Certain pathologists of the modern school are not willing to consider hypertrophy as a physiological process. It is regarded rather as evidence of disease. Krehl, for instance, claims it as certain that hypertrophy of the heart "carries with it in its development the germ of death, and this fact distinguishes it absolutely from the usual work-hypertrophy of the skeletal muscles." Nobody, he says, considers the strong biceps of a gymnast as dangerous, "but what experienced physician would venture to consider an hypertrophied heart healthy, no matter how well it does its work at the time? The great mass of men who have this hypertrophy succumb to insufficiency." Further, he maintains that a clinician may cite a number of cases of improvement under proper treatment, but none in which the heart symptoms entirely disappear. Krehl always finds anatomical evidence of disease, usually in some of the processes of arteriosclerosis in the hypertrophied muscle of the heart. Von Basch is not able to find anything compensatory in hypertrophy but regards the increase in size under long pressure from within as a kind of "accommodation hypertrophy" which rather hinders than helps a valve lesion by increasing the resistance in the heart chambers. Every physician knows, he says, that along with extreme hypertrophy goes dyspnœa, and it is only in the hypertrophies of moderate degree that the heart lesion is masked or concealed.

Legroux, too, declares that hypertrophy may not occur without the intervention of an inflammatory process.

Von Noorden distinguishes the hypertrophy of the heart in diabetes as a welcome complication; for of itself alone it is never the cause of any distressing symptoms, but he admits at the same time that the muscular tissue of hypertrophy shows a greater tendency to subsequent exhaustion and weakness than does the tissue of the normal heart. Frerichs showed long ago that heart failure, entirely independent of coma, is a not infrequent cause of death in diabetes. The final fatal failure follows immediately some unusual exertion or emotion. We are compelled therefore to admit that the disturbances which lead to hypertrophy of the heart may depend upon myocarditis and that this myocarditis may advance, to lead finally to destruction of the heart. Finally, it may not even be maintained that voluntary muscles do not suffer under continued use. Allbutt finds that the file cutters of Sheffield, whose avocation compels them to use their arms continuously in rapid flexion, show at first a great enlargement of the biceps, but after a few years this muscle wastes and falls far below its normal size.

The prognosis in all cases really depends entirely upon the condition of the heart itself. So long as the hypertrophy compensates the lesion, no matter what its character, the prognosis is good. Any evil effects of excessive hypertrophy may be easily counteracted, as a rule. The prognosis becomes bad, however, when the hypertrophy begins to give way, when compensation is broken, and when symptoms of dilatation indicate the stage of degeneration.

Grave cases of heart failure are marked by gastric catarrh, more frequently by atonic dyspepsia, later by icterus. There is often absolute anorexia, the tongue is coated and the breath is bad. These are bad signs, not only in themselves, but because they interfere with the proper medication.

The prognosis depends next upon the cause of the condition. The enlargement of the heart which occurs in pregnancy is of no gravity. The hypertrophy which occurs under the abuse of the milder stimulants, tobacco, tea, and coffee, usually disappears entirely with restraint of the indulgence. The same prognosis may be made in cases of abuse or perversion in venery. Much more grave is the outlook in Bright's disease, but here, too, cases differ according to the form, extent, and duration of the disease. In a general way it may be said that the condition of the heart is the best gauge of the gravity of the disease. Uræmia sets in rapidly when the heart fails. Uræmia is always a bad sign.

So something of the prognosis may be determined by the symp-

toms. Uræmia is itself a worse sign than dropsy, cyanosis is a worse sign than dyspnœa. Tachycardia, which indicates dilatation, is a worse sign than a rigid artery, which indicates arteriosclerosis. A patient may be bloated with dropsy and be afflicted with such dyspnœa as to be unable to lie down, and yet recover. But the patient who suffers from the convulsions of uræmia, or who is blue with cyanosis, is in a very bad way indeed. The prognosis depends also upon the age, the sex, and the habits of life. The cases of Bauer furnish the following results:

(*a*) Idiopathic hypertrophy at maturity up to the age of 50, 76 cases, 27 fatal. Of the fatal cases, 14 were between 22 and 40, and 11 between 40 and 50 years. Of all the fatal cases not a single one belonged to the female sex; 49 cases left the hospital improved.

(*b*) Idiopathic hypertrophy in advanced life, 70 cases, of which 23 (8 men and 15 women) died. Of the fatal cases in women, 7 were over 70 and only 2 were less than 60. Of the fatal cases in men, only 3 were over 65. Of the 47 cases improved, 27 were male and 20 female, and of the female 11 were over 60.

These numbers show that the alcohol heart in the majority of cases belongs to younger men and very rarely to women, and that in the senile changes of advanced life, which play a more or less important rôle, the female sex suffers about the same as the male.

Finally a factor of the utmost importance is the effect of heart stimulants, especially of digitalis. Brilliant results are often achieved in apparently desperate cases under the proper use of the heart stimulants. Cases of extreme dilatation have been rescued in this way. The dropsy disappears, the dyspnœa is relieved, the quantity of urine increases, the appetite improves, sleep is secured, and, in short, all the bad symptoms of failure on the part of the heart are driven away, often in the course of a few days or weeks, under this treatment. Thus the period of danger is bridged over and the individual may be restored for a long time to a life of usefulness. When digitalis or other cardiac stimulant ceases to tone the heart, the outlook becomes bad, and it may be said in a general way that the outlook is never so good when appeal has to be made to other stimulants than digitalis. The prognosis is never good when the stomach is bad or when it becomes bad under the treatment. But no one need ever despair of a case, however grave, until the proper remedies fail.

Prophylaxis.

Much can be done in the way of prophylaxis. The physician should acquaint the patient with the exact character of the disease

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that he may arrange his life accordingly. The patient should be made familiar with the fact that he need have no fear so long as compensation is perfect, but that he must not break this compensation by imprudence in his habits, by excessive work, and above all things by the strain which is put upon the heart by worry. Two things must be guarded against: on the one hand fear, especially the fear of sudden death, which keeps the heart under strain. The patient is to be assured that he may live a long and useful life, that he may finally die of old age. On the other hand, he must guard against indifference or heedlessness with regard to excesses and exposures. More patients suffer in this way than from fear. This statement applies to both sexes; to young men in student life, in the overstrain of physical exercises, in gymnastics and sports, boat-rowing, bicycle riding, baseball, football, cricket, etc.; to young women in society life in excesses in the ball-room, in the strain of social agonies, and later in domestic worries.

A young woman with heart disease should never marry; having married, she should not become pregnant; having become pregnant, special attention should be paid to the action of the kidneys and the habits of life, and labor should be abbreviated as much as possible. Stoffela has shown how pregnancy aggravates the danger of heart disease, and not only that, but how it entails immediate danger to life. Death occurs in at least one-fifth of the cases soon after delivery, usually from œdema of the lungs.

An individual affected with hypertrophy of the heart may be considered only apparently healthy. An hypertrophied heart will always break down sooner than a sound heart. The individual, therefore, who would conserve the hypertrophy as long as possible will pay greater attention to detail in his habits of life. He will dress with more care with reference to the changes in the weather. When the patient's means afford it let him seek a warm climate in the winter. He will guard his diet, eating simply and substantially at breakfast and dinner, but making his supper light. In his food he will be careful as to quality as well as to quantity. He will see to it that the bowels are moved regularly and that the kidneys act freely. He will live in a light, warm, well-ventilated room; he will sleep lightly but warmly clad, and will secure an abundance of fresh air at night.

This patient also will stand guard over his emotions. He will exercise self-control; he will not allow himself to be betrayed into a fit of anger; he will put away sources of annoyance and worry; he will accustom himself to self-denial. All these things and many more may, perhaps, go without saying. The observance of these

precepts does not make life less worth living; on the contrary, they have been inculcated by philosophers from all time as the best modes of life for all men. Socrates said long ago that he who had the least wants approaches nearest to the gods, and Goethe declared that a man would become miserable when every wish was gratified.

The treatment must have reference to the condition of the heart, to the cause of the hypertrophy, and must be addressed to the relief of individual symptoms.

Treatment.

The effects of excessive hypertrophy are most readily relieved by the application of cold. In the interesting experiment made by the physiologist it is seen that when a piece of ice is held on a plate of glass immediately above a freshly exsected heart from a cold-blooded animal, the beats of the heart are quickly reduced in frequency and force. When at last they stop altogether the heart is placed upon the glass and held over an alcohol lamp; the beats begin again and are soon restored under the stimulus of heat nearly to their natural strength. Tumultuous and violent actions of the heart are thus restrained by the external application of cold. When the heart beats violently the vessels throb in the neck, the eyes are suffused, the head aches. This excessive action may be restrained by the application of an ice-bag or by compresses wrung out of cold water. More sustained effects are secured by the wearing of a hollow tin shield filled with cold water. A broad, thin flask of ice water may be carried in an inside breast pocket of the vest. Less excessive action of the heart may be restrained by the use of the bromides, especially the sodium bromide, in dose of 20 to 40 grs. in half a glass to a glass of Seltzer water or soda water from a siphon bottle.

So soon as signs of degeneration ensue, that is, so soon as any dilatation has taken place and the compensation is broken, it is time to interfere.

The first interference is the injunction of rest. The heart can recover its tone only when it is allowed to do less work. If the signs of dilatation are acute, the individual should remain in bed. Nothing so quickly relieves dyspnœa, palpitation, and heart failure, as the recumbent or semi-recumbent posture, and nothing so quickly aggravates or intensifies these symptoms as continued work.

With the subsidence of the acute symptoms under rest, that is, the recovery of tone as manifest in the improvement in the condition of the pulse and in the increase in the quantity of urine, steps may be taken to tone the heart muscle by exercise. As stated elsewhere it was a happy thought, the idea that the heart muscle could be toned

like any other muscle by the proper exercise. But the amount and character of the exercise must be nicely graded to the individual case. The exercise should never be carried to the point of fatigue. The supervention of palpitation, of short breathing, of precordial distress these are signs of overstrain. It is easy to exhaust the heart by exercise. In all cases exercise in the open air is best. The gymnasium of the Greeks had no roof. But the exercise may begin at first in the room, perhaps most profitably, certainly most pleasurably, with some of the machine apparatus, as in rowing boats, riding artificial horses, etc. When the heart can stand it, the best exercise is the climbing of mountains. Shady mountain resorts are finest where stops may be made from time to time to rest, and if from these stopping places a pleasing prospect opens out, so much the better for the patient. The patient comes to these things gradually, of course. The exercise may be at first passive and then active; under all circumstances the patient stops this side of fatigue.

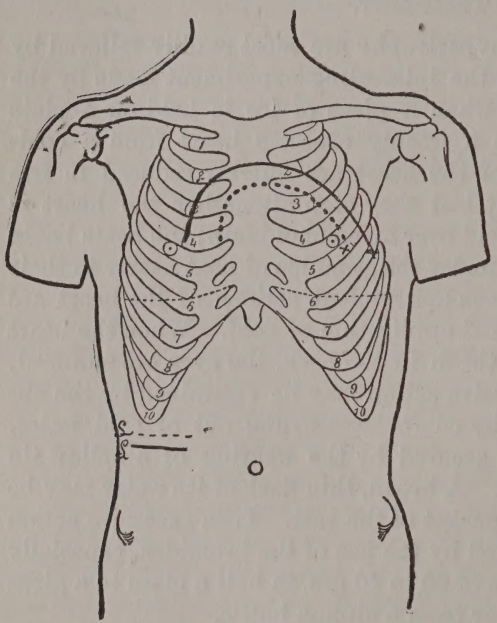

FIG. 5.—The Outlines of the Heart and Liver as Reduced by Exercise. (Schott.) The continuous line represents the dimensions of the heart before, the dotted line the reduction accomplished by exercise. The straight and broken lines in the hypochondrium represent the outlines of the liver under stasis and the retraction effected by exercise.

These good effects of a properly graded exercise render apparent the evils of the opposite treatment by total inactivity. Less than a quarter of a century ago absolute rest was enjoined upon all patients affected with heart disease. Subsequent experience enables us to draw the lines in this regard. Rest is necessary in acute myocarditis, and in the stage of acute dilatation from whatever cause, but chronic degenerations are best overcome and muscular tissue is best developed by systematic exercise. Too much rest favors the processes of degeneration, favors the granulo-fatty degeneration, the hyaline and sclerotic change under which the active protoplasm of healthy muscle is reduced to inert matter.

The next thing is the diet. The best food is milk. Acute heart

disease as a sequel to scarlatina or other infectious disease is often prevented altogether by an exclusive milk diet. Milk is both food and drink. It is converted quickest and with the least effort on the part of the digestive organs into the blood, and is appropriated soonest and most completely by the tissues. Milk is also a diuretic. It flushes the kidneys and thus eliminates toxines. Under a milk diet the nutrition improves, sometimes the dyspnœa and dropsy subside. The only contra-indication, aside from some idiosyncrasy, is offered in certain cases of plethora, where it is not advisable to increase the quantity of blood. In fact, Oertel advised a great reduction in the quantity of fluids as the first principle in the treatment of a weak heart. The best criterion to go by in these cases is the quantity of urine. Where the urine is abundant and clear, milk may be given freely. For the rest, as regards diet, it should be simple and nutritious. The supper should be very light.

So long as the hypertrophy is compensatory, interference is always meddlesome and is often mischievous. Irreparable damage is often inflicted by routine stimulation in all cases of heart disease. The heart which is doing its work well is brought by overstimulation into a state of rapid exhaustion. When the heart compensates the lesions it should be let alone. Thus a nose-bleed is often salutary. It relieves pressure in the head and sometimes in this way prevents the danger of rupture of an atheromatous vessel in the brain. It is a natural venesection. Nose-bleed should, therefore, be checked only in the presence of signs of exhaustion. A diarrhœa and profuse sweat may relieve surcharged vessels in the same way. The physician will regulate his therapy accordingly, favoring rather than preventing these modes of elimination.

Anything like a profuse or exhausting nose-bleed may usually be stopped at once by touching the septum near the orifice with a fresh saturated solution of chromic acid. Colliquative diarrhœa may be controlled by bismuth grs. x.–xx. every two to four hours, and profuse sweats by a solution of atropine gr. i. – ℥ i. of which gtt. iij. may be given three or four times a day; or often better by strychnine solution in the same strength, in doses of gtt. x. at the same intervals.

Where the processes of arteriosclerosis are pronounced, as indicated by the condition of the radial or temporal pulse, by the form of kidney disease, by any of the signs of intermittent closure of vessels, by the indications of thrombosis in the cerebral arteries, etc., the potassium or preferably the sodium iodide should be regularly administered. The drug may be dissolved in equal parts of peppermint water, and the dose of this preparation should range from five to ten drops in a wineglass of milk before meals. There is in this

preparation no incompatibility with digitalis or other form of heart stimulant, and the iodides may be given regularly while the digitalis is continued, once or twice a day, or oftener as may be, for a period of weeks and months.

Anæmic patients may be greatly benefited by the use of some ferruginous mineral water, especially by the combination of iron with arsenic, as existing in Roncegno water and Levico water, preparations that may be taken in the dose of a teaspoonful, dessertspoonful, or tablespoonful in a wineglass of water after meals. Arsenic in minute dose improves the processes of assimilation we say, in concealing real ignorance under words. It may be that it acts by some antidotal or antimycotic properties. At any rate, the clinical fact is established and accepted. Where for any reasons it is advisable not to give arsenic, the iron may be administered in the form of the tincture along with the tincture of digitalis, or a teaspoonful of the saccharated carbonate of iron may be taken after each meal. Arsenic is contraindicated in fat people and in fatty heart.

The heart may be now additionally sustained by the use of drugs. Digitalis is the finest of all the heart stimulants, but the remedy must be used with judgment. The dose should be regulated in quantity and frequency to the wants of the individual case. A mild case is benefited by the use of the tincture of digitalis in dose of five to ten drops every three or four hours. In a more severe case the infusion may be preferred, in a dose of a teaspoonful to a tablespoonful every three to four hours. Inasmuch as neither alcohol nor water alone dissolves all the essential elements, preference may be had at times for digitalis in powder in the dose of a grain to a grain and a half, every two, four, or six hours. See also the section on the treatment of Valve Lesions.

Where digitalis disagrees with the stomach, it may be substituted by strophanthus or sparteine. These rivals are, however, far behind. The tincture of strophanthus is administered in the same dose as the tincture of digitalis. The sulphate of sparteine is given in the same dose as the sulphate of morphine.

For steady and continuous support, no remedy equals strychnine. The tincture of nux vomica, drops ten to twenty, is a good preparation, or the strychnine itself may be given in the form of the sulphate or nitrate, in a grain to the ounce solution, of which the dose may be ten drops three times a day. Quicker effects may be had from the subcutaneous injection of the sodium benzoate of caffeine, in the dose of three to five grains. Nitroglycerin acts kindly in cases of kidney disease, in that it diminishes the blood pressure in the periphery.

Constipation is best relieved by some of the vegetable pre-

parations, especially by compounds containing small quantities of strychnine, which stimulates the sluggish peristalsis, while it at the same time improves the tone of the heart. A pill in common use is the combination of aloin, belladonna, and strychnine, one of which may be taken after each meal. In the presence of much flatulency, which is a common and distressing complication of heart disease, this pill may be substituted by a preparation which contains asafœtida, as in the compound pill of galbanum in the composition of which the asafœtida may be increased if necessary.

A failing appetite may be assisted by exercise in the open air or by a powder of basic orexin, once daily, at ten A.M., 0.3 (gr. v.) in a meat soup, which is often of excellent service when the appetite does not recur during the intervals of digitalis treatment.

Palpitation is best relieved by rest and is prevented by the judicious use of digitalis. The attack itself may be cut short by the administration of a stimulant, as a teaspoonful of good cognac, of an anti-acid, as a ten to fifteen grain dose of carbonate of soda, or of ten to fifteen grains of the salicylate of soda. A hot foot bath and a mustard plaster over the region of the heart are home remedies not to be despised in securing quick relief of a severe attack.

Dyspnœa is best relieved by rest and by stimulation of the heart with heart tonics, especially by digitalis. When the attack is so severe as to amount to cardiac asthma, it may be treated by the same remedies as for the relief of palpitation, more especially by the diffusible stimulants, brandy, ether, and camphor. Sometimes a teaspoonful of a solution of chloral hydrate (two or three grains to a drachm of peppermint water) proves of great service. A teaspoonful of brandy or whiskey should always be given with the chloral, or where this remedy is oftener used it should be preceded by the administration of dilute hydrochloric acid, ten to fifteen drops in a wineglass of water. When the attack is obstinate it may be cut short by the subcutaneous injection of morphine. For the relief of the severer attacks of dyspnœa, morphine is the best remedy. It is also sometimes the best hypnotic, but it must be prescribed with care, first in the tentative dose of 0.005, gradually increasing to 0.01, one-sixth of one grain. In the relief of œdema diuretin gr. xv. ter die in soda water from a siphon bottle may be tried, and should necessity arise the hydrops and anasarca may be relieved mechanically, *i.e.*, by incisions.

Precordial pain calls for rest, cardiac tonics, strychnine, digitalis, and counter-irritation in the form of a mustard plaster. On the other hand, in some cases the application of cold is grateful. Where the pain is severe resort must be had to morphine.

Anything like an attack of angina may be cut short by the use of the nitrites, especially by the amyl nitrite. As stated elsewhere, patients thus afflicted may carry with them glass pearls which they may break in a handkerchief and inhale the fumes. Sometimes large quantities must be taken to secure the desired effect. Balfour speaks of the great freedom with which amyl nitrite may be used by certain individuals, and narrates the case of a friend who suffered much with angina in connection with aortic regurgitation, and who got relief by inhaling the remedy during the day, but not content with this quantity he "used to soak his pocket handkerchief in the amyl and go to sleep with it lying on his face, without any ill results." More sustained effects are secured by the use of nitroglycerin in the dose of one, two, or three drops of a one-per-cent. solution, one to three times a day. This remedy may be given also in the form of tablets with chocolate, each containing the $\frac{1}{100}$ of a grain of the drug.

The heart complications of gout are best counteracted by the proper regulation of the food, gradation of exercise, and exhibition of the salicylates, preferably the salicylate of soda. Salol should be avoided in the presence of any kidney troubles. It is often remarked that when the gout localizes itself the complication on the part of the heart disappears. Balfour thinks that this substitution could be reached and the same good accomplished by the use of colchicum in doses of five to twenty drops of the tincture.

Sleep is best secured by trional in the dose of grs. xv.–xx. in a cup of hot milk or hot tea at bed-time. See also the treatment of Myocarditis and of Chronic Valvular Disease.

Myocarditis.

Myocarditis (μῦς, muscle; καρδία, heart)—infection (inflammation) of the substance, parenchyma or wall, of the heart.

The term was first used by Sobernheim (1837). At the beginning of the present century Corvisart described the condition as a carditis, *i.e.*, as a phlegmasia, which he believed affected the entire heart in all its parts, muscle, membranes, and vessels. Kreysig, Andral, Bouillaud, Rokitansky considered the carditis as an inflammation beginning in the muscle; Bristowe, Friedreich, Bard, and Phillipe located the affection in the interstitial tissue and called it an interstitial myocarditis; Duplaix, Debove, Haushalter, fixed the point of departure in the vessels, as a peri-arteritis and an extension by proliferation of the connective tissue from the vessels. In elaboration of this view, Martin, Huchard, and Weber derived the inflammation in the first place from the intima as an endarteritis.

Huchard boldly designates myocarditis an arteriosclerosis of the heart.

The conception of myocarditis varies not only with regard to the tissue involved but also as to the character of the affection. Thus the term is limited at times to inflammations proper which may terminate in resolution or in suppuration, and again is extended to include all the various degenerations. As it is at the present time difficult to draw the line between chronic inflammations and degenerations, or as it is admitted that degenerations are expressions of chronic inflammations, myocarditis is still studied rather from the standpoint of acute, subacute, and chronic inflammation, localized or diffuse, understanding by this term a process which results mainly from infection.

From this standpoint the study of the heart is much simplified and the character of the lesion, whether parenchymatous, interstitial, or vascular, is referred to a common cause, to wit, to a poisoning of the blood. Myocarditis is, therefore, a secondary affection, a consequence of other and for the most part infectious diseases.

Acute inflammation of the heart was first seen in the course of or as a sequel to typhoid fever. Louis described the condition which resulted in softening. Andral, Günzburg, Wunderlich, and Stokes described the loss of consistence as so great at times as to reduce the heart to the condition of a wet cloth. In fact, the color was said to be like that of a dead leaf (*feuille morte*). Stokes saw a case in which the softening was so great "that when the heart was grasped by the great vessels and held with the apex pointing upward it fell down over the hand, covering it like the cap of a mushroom." Romberg very properly suggests that too much attention is paid to the mere condition of the softening, which in muscle like the heart is naturally found at the time of autopsy in the most different degrees of post-mortem rigidity.

Etiology.

The myocarditis of typhoid fever has always served as a prototype in the description of infection of the heart muscle. Stokes has made us familiar with the symptomatology of the complication. The frequency of sudden death from heart failure in diphtheria soon attracted attention to the affection of the heart muscle in this disease. Rosenbach described a parenchymatous degeneration, Birch-Hirschfeld interstitial proliferations of high degree. Hereupon Leyden published a series of cases, and Romberg described in detail the various processes of degeneration. Soon it was seen that diphtheria is one of the most frequent causes of infectious myocarditis, and that

the greatest danger of the disease exists in this complication. Diphtheria produces myocarditis much more frequently than endo- or pericarditis, and while inflammations of these various tissues are often found associated, myocarditis sometimes exists alone.

Diphtheritic myocarditis usually sets in late. It begins between the sixth and ninth days, reaches its acme at the end of the second or beginning of the third week, and may then continue with undiminished intensity far into convalescence.

The process is therefore post-diphtheritic. It is rarely seen in children under the age of six years, chiefly on account of the rapid mortality of diphtheria at this age, at least before the present period of specific therapy. It occurs most frequently between the ages of six and fourteen years, more commonly among boys, but by no means infrequently in adults.

Leyden first pointed out the changes in the heart muscle which occur in the course of scarlet fever, and Romberg remarked upon the early period in which the heart may be affected in this disease. Thus fatty degeneration of the musculature is sometimes found as marked in the first days of this disease as in other affections only in later stages. Interstitial myocarditis may begin as early as the fourth day, but increases in severity up to the middle of the second week.

Myocarditis has also been observed in the course of cerebro-spinal meningitis, variola, erysipelas, malaria, rheumatism, and especially frequently in the various forms of septicæmia. In fact, the micro-organisms which most frequently produce myocarditis are the streptococcus and staphylococcus of pus. When these micro-organisms are introduced directly into the circulation, as in the experiments of Ribbert, with fragments of potato containing pure cultures, myocarditis occurred more frequently than inflammation of the membranes of the heart. But even trivial infections may lead to myocarditis. Thus Kelle declares that he has lately seen in the Leipsic clinic several cases of grave acute myocarditis in the course of simple quinsy.

That the heart muscle is sometimes severely affected by the poison of influenza is indicated by the weakness of the heart which shows itself entirely independent of fever. Thus there are cases in which the activity of the heart remains intact during the highest fevers, while there are others in which the signs of heart weakness with systolic murmurs occur even in low fevers. Burney Yeo remarked upon the occurrence of heart weakness of grave character attended with signs of extreme stasis, which disappeared only after long rest in bed and protracted roborant treatment of the heart, as

sequels to influenza so mild as to have passed almost unnoticed by the patient. Sansom saw in thirty cases of influenza irregular action of the heart which was attended with all kinds of nervous disturbance, paresis, paræsthesia, exophthalmus, struma ptosis, tinnitus, *vertigo ab aure læsa*, precordial anxiety, etc. The effect of influenza in aggravating chronic disease, often to a fatal termination, is largely due to the degradation of the heart (Leube). The total inefficacy of digitalis and other heart stimulants in lifting the tone of the heart throughout the course of the disease in certain cases, or during the crisis of pneumonia, finds its explanation in the grave toxic influence of these affections upon the muscle of the heart.

As for pneumonia the condition of the heart muscle is the actual gauge of the gravity of the disease. Heart failure is the chief cause of death in the pneumonia of alcoholism and of old age.

Tuberculosis of the myocardium is rare, though the heart muscle wastes with other organs in the process of phthisis. Observations of the occurrence of embolic tubercles in the heart are not frequent in man, but two cases have been reported, one by Preusze and one by Lungwitz, in the cow. Ströze found a number of nodules of tuberculous nature. In the central part of one tumor caseous matter was found in which tubercle bacilli were demonstrated under the microscope.

Gonorrhœa is the latest infection known to produce myocarditis. In this case, also, the relationship was first established by Leyden, whose publication was soon followed by the fine and full demonstrations of Councilman. In some cases the originating malady, of whatever character, may have long since disappeared or may have been so slight as to have passed unnoticed. Sometimes it was called "a cold."

How the infections act in the production of myocarditis has not been definitely determined. In certain cases the micro-organisms have been found in the substance of the heart. Such demonstrations have been made in septicæmia with the discovery of pyogenic micro-organisms (Litten, Ziegler), and in gonorrhœa with the disclosure of the gonococcus (Leyden, Councilman). Landouzy and Siredey discovered the typhoid bacillus in the heart, and, more to the purpose, Chantemesse and Vidal found it in the heart in a case of typhoid myocarditis. But, on the other hand, it is only fair to state that Lyon and Gilbert were not able in their experiments with the typhoid bacillus to produce a myocarditis.

Sometimes the myocarditis results from the action of micro-organisms not directly connected with the production of the original disease, as from the various pyogenic micro-organisms introduced in the course of other diseases to constitute the process of secondary or

mixed infection. Thus the inflammation of the myocardium which occurs in pneumonia is not always to be referred to the diplococcus of pneumonia (though recent investigations have shown it to be very diffuse), but to associated pyogenic micro-organisms which are ubiquitous. Finally, among the more masked infections, syphilis affects the heart, but this process is usually considered separately.

In many cases the myocarditis does not seem to result from the direct presence of the micro-organisms. In the studies of Kockel bacteriological examinations furnished only negative results. It is noticed in many cases that the myocarditis occurs, as in typhoid fever, late in the history of the infection, also that it shows itself in connection with diseases, such as diphtheria, caused by micro-organisms which do not penetrate to the blood. It is seen further that affection of other voluntary muscle, which shows itself as paralysis, may develop at the same time with the myocarditis. This is especially apt to be the case in diphtheria and sometimes in dysentery. The paralysis of the voluntary muscles which occurs in diphtheria is nearly universally attributed to the action of a toxalbumin. So that at the present time the tendency is to ascribe myocarditis not so much to the direct invasion of micro-organisms as to the action of the specific toxins or toxalbumins which they develop.

The objection that it is necessary to demonstrate bacteria in the affected region to prove an infectious origin is no longer valid. It is known that certain poisons, aconite, digitalis, muscarine, veratrine, distinctly affect the action of the heart, and it is known that certain toxins exercise this same effect. Thus Klein found that the injection of the toxins of micro-organisms produce rapid granulo-fatty degeneration in the liver, kidney, and heart, and Brault and Charrin observed the various changes of myocarditis, acute, subacute, and sclerotic, after the introduction of filtered cultures of the bacillus pyocyaneus.

Alcohol leads directly to degeneration of the heart muscle through over-stimulation and exhaustion, and indirectly through toxæmia. The heart is overburdened further by the increase of blood pressure. The toxic effects are observed soonest and most marked in cases where alcohol is taken in concentrated form as in brandy, whiskey, rum, bitters, and still more in cases where the alcohol is impure. The increase of pressure is observed especially in cases where the alcohol is taken in more dilute form, as in beer and wine. Billings showed that the mortality of dealers in liquor, aged from twenty-five to sixty years, was very much greater than that of the men generally. In a thousand cases, one hundred and twenty men generally and one hundred and forty liquor dealers died from diseases of the circulatory system alone.

Chronic Myocarditis.

Diffuse degeneration of the heart muscle ensues in the course of time under powerful muscular effort. Overstrain of the heart is produced in certain vocations, as in porters, shovellers, boiler-makers, founders, steam-pipe fitters, etc. Jürgensen noticed the degeneration of the heart muscle which occurred in the workers in vineyards who had to climb hills with heavy burdens on their backs. But excesses in this direction are not confined to the working classes. The writer once had a case, which terminated in sudden death, in a cornet player, who was, however, also a great beer drinker. Mountain climbers, gymnasts, bicycle riders, base- and football players, boat rowers, commonly break down prematurely with degeneration of the muscle of the heart. The same effect is produced in the course of time by valve lesions, the evils of which the heart first overcomes by hypertrophy, but subsequently yields to in dilatation and degeneration. Emphysema of the lungs and frequent repeated and long-continued severe attacks of asthma throw extra work upon the heart in the same way, and in the long run may produce the same results.

In obesity the heart is overloaded with fat, which is sometimes insinuated between the muscle fibres to constitute the process known as fatty infiltration. Fatty degeneration occurs later in the history of these cases. Obese patients show signs of heart failure in dyspnœa and early exhaustion. Obesity is nearly always associated with arteriosclerosis. See also under Fatty Heart.

Age shows its effects more especially in the degeneration of the arteries in the process of arteriosclerosis. But age induces the same atrophic and degenerative changes in the heart muscle as in the other muscles and tissues in the body.

Chronic myocarditis depends in most cases upon arteriosclerosis, and any disease which precipitates this process develops the disease of the heart. The chief causes of premature arteriosclerosis are alcoholism, syphilis, Bright's disease, lead-poisoning, and gout. The rôle of these various factors excepting gout is discussed in detail in the sections on Hypertrophy and Sclerotic Endocarditis. The relation of gout is established in "The Gouty Heart," which has become almost a synonym of arteriosclerosis. Schott found that gout, arthritis urica, figured in the production of anomalies of the heart in 54 cases, mostly in the way of changes in the blood-vessels, especially the coronary arteries with their consequences, angina pectoris and dilatation. The attacks on the part of the heart occur especially in persons who suffer with irregular gout without affection of the joints. In this

connection Fothergill cites the case of a dyspeptic old person who once boasted: "I have been a dyspeptic for fifty years! Thank God for it!" "The cause of his pious gratitude was the fact that all his brothers had been cut off by diseases of gouty origin; the dyspeptic alone survived."

The tendency to arteriosclerosis is also distinctly inherited. Schott found that of 750 patients, 212 declared that one or more members of the family were affected with heart disease. Heredity plays, therefore, a much greater rôle in the etiology of chronic heart disease than is indicated in the literature.

Among the chief causes, interest is excited at once by the extraordinarily high percentage of cases which result from emotional disturbance, either long continued or sudden; further by intellectual excesses. Schott found the condition produced in this way in not less than 140 in 505 cases. In 68 of these cases there was a conjoined physical strain.

With all these causes it is therefore not surprising to learn that diseases of the myocardium are much more frequent than diseases of the endocardium in the proportion of 505 to 245.

Disease of the heart occurred further after profuse hemorrhage in 19 cases, in 14 after metrorrhagia in consequence of rapidly following labors. Other cases were due to hemorrhoids, bleeding from the nose, and uterine fibroids.

So it is certain that a more thorough appreciation of the causes which lead up to myocarditis would enable us to recognize the frequency of the condition in its slow, insidious development, and by recognizing it earlier would relieve medicine from some of the opprobrium which rests upon it in connection with the frequent reports of sudden death from "heart-failure."

Morbid Anatomy.

The changes which occur in the muscle of the heart may be localized or diffuse, or may be, as stated, mainly vascular, parenchymatous, or interstitial.

Where the process is localized it is generally more or less disseminated in certain spots or regions in the left ventricle, especially toward the apex or in the papillary muscles, sometimes in the septum, more infrequently in other parts, and much more infrequently in the right ventricle. Myocarditis, as stated, is generally found associated with peri- and endocarditis, in fact with pericarditis in the majority of cases, but myocarditis may exist absolutely alone. Romberg found it frequently in the trabeculæ of the heart without any

involvement even of the endocardium. The infiltration was confined to the interstices between the fibres or penetrated to their interior. Small veins and capillaries, often thickly stuffed with leucocytes, could be recognized between the heart fibres. The localized processes correspond often to domains supplied by branches of the coronary artery, and may show typical infarctions when the vessel is blocked by an embolus or by the process of thrombosis. Sometimes the hemorrhagic infarction is succeeded by a circumscribed necrosis with softening, myomalacia. Destruction of tissue may thus ensue and the weak part may bulge under the blood pressure to produce an aneurysm, or sometimes to suffer rupture. Thus the contents of a circumscribed abscess may be discharged into the endocardium and be disseminated over the body, or into the pericardium to excite purulent pericarditis, or more frequently to result in rupture of the heart with its usually fatal termination. Sometimes the septum is perforated in the same way and intercommunication is established between the ventricles. More favorable results are resolution, which may take place in the stage of infiltration, or absorption of pus after the stage of suppuration with the formation of cicatrices, and the substitution of the muscular by cicatricial tissue. With this substitution the power of the heart is directly lessened; especially is its reserve force diminished. The condensation and retraction which this tissue always undergoes affect the heart but little practically because of its situation. But if the process be situated in the neighborhood of the orifices of the great vessels, it may eventuate in stenosis with its consequences, or if the tissue yields to pressure, in chronic partial aneurysm of the heart. A most favorable result is the process of encapsulation or calcification which sometimes ensues. The best examples of strictly circumscribed or localized affections are found, as stated, in connection with arteriosclerosis as it affects branches of the coronary arteries. Strictly localized processes also are characteristic of syphilis, which may develop gummata or nodules and leave fibroid cicatrices in the heart muscle. In arteriosclerosis the infiltration is at first strictly perivascular.

Diffuse degeneration may be limited to certain parts, or may affect the whole heart. The process is especially distinguished by the fact that the muscle fibres become opaque, the striæ indistinct, and the muscle tissue is substituted by granules of fat. The color may be changed to gray or yellow, sometimes, with the accumulation of pigment matter, to a darker hue. The tissue is lax and friable. Sometimes individual fibres show evidences of waxy degeneration. Fatty degeneration is especially marked in the myocarditis which occurs in the course of diphtheria, where waxy degeneration is uncommon.

The fragility of the muscular tissue is shown in the cross rupturing of fibres, a striking condition which has been observed especially in the hearts of typhoid fever. These cross tears are situated usually at the seat of the cement substance. A similar process is sometimes described as a fragmentation of the cells of the heart muscle, which has been found in various conditions and has met with various interpretations. Dunin opposes the German view that fragmentation is due almost exclusively to mechanical cause, excessive tension in the death agony, and defends the chemical view advocated by the French and Polish authors, supporting his opinion by citing two cases in which he found a species of bacillus, probably the Bacterium coli commune, in the heart muscle. Unfortunately cultures were not made. Among the finer changes may be further noticed alterations in the nuclei in the form of elongations and enlargements.

The diffuse process is typically shown in pericarditis, which involves a deep zone of peripheric heart tissue underlying the pericardium. Still more extensive are the processes observed in the course of the virulent infections, diphtheria, septicæmia, etc. Typhoid fever may show both localized and diffuse myocarditis. The complication is, in fact, more common than is usually believed. Thus Stein saw parenchymatous changes in the heart fourteen times in 40 cases. Zenker found in 21 cases granular infiltration of individual fibres in 5, extensive granular degeneration in 2, indistinct striation in 2. Hoffmann made the most extensive investigations. The heart tissue was found normal or but little changed in 56 of 150 cases; there was slight granulation in 39, marked granulation in 46 cases. Wagner found albuminoid infiltration in 59 cases, fatty degeneration in only 9 cases.

Where the poison expends its force upon the vessels, it leaves distinct alterations in the lesions of arteriosclerosis. Thus Huchard found with Weber atheromatous lesions in every one of forty-four cases. These lesions were most marked in the left coronary artery thirty-one times, in the posterior coronary artery seven times, and in both branches equally six times. The anterior artery shows the most frequent change because it supplies the left ventricle, which has the most work to do. Hayem found in the myocarditis of typhoid fever a preference of attack of the small branches of the pericardium and in the musculature of the heart itself, while the chief branches were spared. The affection begins with the thickening of the intima and a reduction of the lumen of the vessel.

The parenchymatous change was first studied by Virchow, who described the process of albuminoid granulation and fatty degeneration. Ricord made a contribution on the chronic parenchymatous

degeneration found in syphilis. Zenker followed with his well-known exposition of the waxy degeneration. In his studies of gonorrhœal myocarditis Councilman found the muscle fibres in various changes of degeneration. In the slightest degree they were somewhat swollen, the nuclei were absent, and there was much vacuolation. The changes ranged from this slight degree up to total necrosis of the muscle fibres and entire substitution, in areas, of purulent infiltration and necrosis for the normal tissue of the heart.

Suppurative myocarditis has been known for a long time. Galen described it as a malady of the gladiators. Benevenus was the first to discover an abscess in the walls of the heart. Bonetus in his Sepulcretum cited a number of such cases. Laennec saw only one case. Meckel reported the case of a man aged fifty who died of pericarditis, and in whom the muscular fibres of the heart were found infiltrated with pus.

Whether or not the parenchymatous changes are preceded by changes in the vessels is a matter of dispute of no practical importance. The majority of observers, according to Kelle, connect the destruction of muscle fibres with changes in the arteries, deriving the change in the muscle from the defective blood supply. But the view which is more in accord with modern ideas and which is more likely to prevail, is that advocated by Köster, who considered the arteritis and myocarditis as consequences of a common cause, which cause was probably always some infection. It is certainly acknowledged on all sides that myocarditis, especially a chronic myocarditis, does not necessarily imply any evidence of disease of the vessels. Coronary sclerosis is by no means a frequent affection, while myocarditis is so common, contrary to the view that is generally entertained, that the pathologists no longer show all their cases for fear, as they say, of wearying their students with monotony. The point is simply that the poison of the blood sometimes exercises its toxic effect first upon the vessels to produce an arteriosclerosis with subsequent necrosis and possibly purulent inflammation of the muscle of the heart, but more frequently expends its force directly upon the muscular tissue of the heart to produce parenchymatous and interstitial myocarditis.

The changes in the interstitial tissues are equally important. The proliferation may be so great as to justify the term interstitial myocarditis. Interstitial change is especially significant on account of its sequels, as all hyperplastic tissue must subsequently contract. Depots and masses of cicatricial tissue are thus found scattered throughout the heart. Hayem describes the cicatricial as a distinct stage of parenchymatous inflammation. These changes

occur late, not earlier, for instance, than the third week of typhoid fever. They are more frequent in certain epidemics than others. Romberg maintains that interstitial change occurs always in diphtheria, in typhoid fever in more than half the cases, but only exceptionally in scarlet fever. This author is so convinced of the importance and prominence of the process as to consider it at times as a separate and independent affection. Thus it may be stated, he declares, that the development of cicatrices may no longer be considered as evidence simply of sclerosis of the coronary arteries, but may be due solely to interstitial myocarditis.

Of these various processes, the fatty degeneration is most characteristic of diphtheria, and is, as stated, the most frequent cause of death in the earlier periods of this disease. Cicatricial change begins later, in the third week, but may become extensive in the course of the fourth week. Parenchymatous degeneration is most frequent in typhoid fever, and suppurative change in the processes of septico-pyæmia and glanders.

Hitherto have been noticed only the changes which affect the muscular tissue, the connective tissue, and the vessels, but the nerve elements themselves do not escape. Since the demonstration that the heart ganglia have to do with sensation and not with motion, any change which affects them has assumed secondary value. Nevertheless, the perception of sensation has much to do with the regulation of the force of the heart, as it is through the nervous system of the heart that is discharged the depressing influence upon the vaso-motor system which reduces the action of the heart and thus lessens its waste.

Changes in the ganglia were remarked in diphtheria by Ivanowski, who found the cells swollen and opaque, so that the nuclei could scarcely be distinguished. Granulation tissue was observed in the connective tissue of the ganglia and embedded between the nerve cells. Putjatin found hyperæmia and granulation tissue in the nerve ganglia with hyperplasia of the connective tissue and subsequent degeneration of the ganglia into pigment matter. Wasileff discovered a similar condition in the nervous system of the heart in hydrophobia. Uskoff saw thickening of the capsule and proliferation of the nuclei. Winogradow, in two cases of chloroform-poisoning, found the nerve cells degenerated in high degree, less translucent, and strewn with granules which sometimes filled the protoplasm and concealed the nuclei. The same author found also in infectious pneumonia parenchymatous inflammation of the ganglion cells of the heart. Ott found the protoplasm cloudy, subsequently the nuclei increased with the formation of connective tissue about the ganglion cells, obliterating the nuclei; there was also an abundant deposit of fat

granules. These changes in the ganglia were associated with changes in the musculature of the heart, marked by fatty degeneration, hyperplasia of its connective tissue, with brown atrophy. It is interesting to know of these cases that there could not be discovered in life any evidence in the heart, as regarded the strength, frequency, or rhythm of the action, of the histological change in the ganglia. Fraentzel had for years considered it dubious if any microscopical examinations of the ganglia would explain the irregularities of the heart, and found the results of these examinations confirmatory of his opinion. This statement, however, may not be taken as final, as the same thing has been said of changes in the heart muscle. It is not probable that all the heart ganglia were studied in any case. Romberg found perineuritis in almost half the cases of typhoid fever and diphtheria, but never in the hearts of scarlatina. According to the investigations of Woolridge, the nerves of the ventricles, which are especially implicated, influence the blood pressure through reflex action, and disease of these nerves probably acts, as stated, injuriously upon the mechanism of the circulation.

In this connection may be mentioned the changes in the heart ganglia which occur under starvation. Statkewitsch found in these cases that the heart ganglia underwent a fatty degeneration in a small degree, but in a large degree experienced a pronounced vacuolization, whereby the nuclei undergo certain changes of form. The ganglia lose thereby their functional activity and probably bring about earlier death from starvation than would follow from degeneration in the other organs.

In no case could it be said that the changes in the heart were produced simply by fever, for the muscle of the heart has been found free or affected in only slight degree in diseases attended with high fever, and, on the other hand, severely involved in cases attended with little or no fever. Thus rheumatism, which is characterized at times by extreme hyperpyrexia, produces far more frequently pericarditis and endocarditis than myocarditis. Diphtheria, which shows no high or long-continued fever, sometimes no fever at all, produces myocarditis of extreme degree, while typhoid fever with its continuous fever, and scarlatina with its high fever, usually show degeneration of the heart muscle in light degree.

Symptoms.

As already stated, the lesions of myocarditis may be latent. This condition may apply, however, only to cases in which the lesions are limited. Any extensive degeneration must show itself in symp-

toms which are sometimes of the gravest character. It makes a difference also whether the disease assumes a slow or rapid course. A process which extends rapidly may take life before there is time for the development of lesions. On the other hand, the heart begets a tolerance in processes which develop very slowly. But, as a rule, it may be said that symptoms and lesions correspond. The symptoms may be summed up under the general term weakness of the heart, and the weakness may show itself as a sudden collapse, heart failure with sudden death, or with some of the numerous signs which indicate gradual and increasing loss of power. Thus the general condition may differ greatly in different cases. Certain patients have the appearance of healthy people, others show signs of grave insufficiency of the heart, and between these extremes is every gradation of appearance and condition.

The first indications of weakness are usually felt in a vague sense of uneasiness, emptiness, or anxiety in the region of the heart. Ordinarily an individual in health is unconscious of the action of the heart; the hypochondriac may feel sensations which really exist through disturbance of innervation. Weakness of the heart makes itself manifest by a feeling of discomfort or distress and by complaints of palpitation and disturbed action, which sooner or later may be appreciated by physical examination. When the process is very gradual, the weakness shows itself in increased irritability, erethismus cordis, in palpitation or increased action which is easily excited by slight physical effort or emotional disturbance. This condition of heart weakness is seen in the earlier stages of tuberculosis, diabetes, Addison's disease, etc., and is especially characteristic of neurasthenia. The increased action may amount to a real tachycardia, and the distress, as stated, to positive pain. In fact, circumscribed, painful sensations in the precordial region, or points painful to pressure from associate intercostal neuralgia, occur, as a rule, in the earlier stages of heart weakness. The fact should be familiar, as at this time the lesion is not organic and the condition is, therefore, entirely amenable to relief. Where the process is distinctly of infectious origin as in pneumonia, gonorrhœa, pyæmia, the implication of the heart may be announced by chilly sensations, which are especially liable to occur in the night and to be attended with or followed by attacks of palpitation and pain in the region of the heart, which may last a quarter to half an hour. These symptoms may show themselves, of course, in the dissemination of any infectious process, and necessarily indicate affection of the heart only when found in connection with other signs.

The weakness of the heart may be apparent in the feebleness of

the apex stroke, which may make no visible impression upon the walls of the chest, and may often, under the most favorable circumstances, as in thin subjects, not be felt at all. The condition becomes still more apparent under auscultation when the heart sounds seem muffled and remote. The weakness is apparent also in the pulse, which is feeble or fluttering and may fade away entirely when the arm is lifted at right angles from the body. This weak action of the pulse often excites anxiety, especially in the later stages of typhoid fever, and most emphatically enjoins absolute rest of the body. Griesinger first called attention to the irregularity of the pulse in typhoid fever, and Traube made a bad prognosis with the sinking of the pulse frequency in diphtheria. Romberg states that he saw in a case of scarlet fever a reduction of frequency from 120 to 52 a few hours before death.

But the pulse is not only weak, it becomes also irregular and intermittent. Nearly every pulse wave may show a different size; a large wave may be followed by one or several small waves, etc. The irregularity is less marked than the inequality, though a number of strokes may succeed each other rapidly to be followed by intervals of various length. The irregularity shows itself more especially in arhythmia in any of its varied forms, sometimes in a bigeminal and alternating pulse, sometimes in change in the character of the sounds of the heart. Huchard emphasized the presence of the peculiar rhythm found in the fœtal heart as the embryo-cardiac rhythm, and Leyden called attention to the galop rhythm in diphtheria as one of the best signs of dilatation of the left ventricle. Various theories have been propounded to explain the galop rhythm of the left ventricle. Sibson, Barr, and Sansom attributed it to a duplication of the first sound resulting from the successive closure of the tricuspid and mitral valves. The contraction of the left ventricle is later than that of the right and is thus separated from it to double the first sound. According to Exchatquet and Johnson the sound is produced by an exaggerated action of the auricle. It is an intensification of the auricular systole. Potain attributes the bruit to a shock of diastolic tension. A more satisfactory explanation is found in the study of the process of contraction in a muscle as it suffers exhaustion. It has always been known that there is no such thing as absolute uniform contraction, but the various fibres which compose a muscle contract in health nearly simultaneously. During the process of exhaustion the synchronism is destroyed and the irregularity of contraction becomes more manifest. Traube and Rosenstein pointed out long ago that the systole is not composed of a single, but of a series of many contractions. In some of the larger lower animals the ventricle succeeds in expelling its con-

tents only after a number of repeated efforts in quick succession. Chauveau finds that the ventricle of the horse empties itself only in two or three successive efforts. According to D'Espine the superadded sound is a duplication or division in this way of the systolic bruit, and this division of the systole into two separate periods gives rise to the peculiar rhythm of the galop.

The weakness in the heart is shown also in its effects. Diminution in the action of the heart is quickly observed in the secretion of urine. The quantity sinks to one-half or one-fourth of the normal amount. Sometimes there is absolute anuria. Albuminuria is almost always present at this time, and in certain diseases attended with rapid collapse of the heart and diminution of urine, the signs of uræmia soon supervene. This condition is observed with especial frequency in cholera, and the so-called cholera typhoid is usually attributed to uræmic intoxication. Renal insufficiency is a precocious and almost constant symptom of myocarditis even in the absence of albuminuria (Huchard).

Difficulty of breathing sets in sooner or later. The patient finds himself short of breath upon the slightest exertion, and sometimes paroxysms of dyspnœa occur spontaneously. This dyspnœa is partly mechanical and partly toxic. The mechanical factor is easily understood in the diminished action of the heart and the dilatation. Dyspnœa is due in all cases to defective oxygenation of the blood and tissues. Glaisher found that at an elevation of 5,500 metres (18,040 feet) violent palpitation set in with an elevation of the pulse and an impeded respiration. . . . At a somewhat greater height the hands and lips became livid, a certain sign of lack of oxygen in the blood; and at an elevation of 7,000 metres (22,970 feet) great dyspnœa set in and he felt sick and wretched. Consciousness was lost at an elevation of 8,200 metres (26,916 feet) (von Liebig). The toxic element is found in the kidneys, and renal asthma has been dignified in the past as a distinct disease.

Bouchard made some curious calculations in this regard. This observer found that in ordinary conditions it requires at least 0.45 to 0.50 c.c. of normal urine per kilogramme to kill an animal. For a man weighing sixty kilogrammes the urotoxic coefficient (that is, the sum of the urotoxin per kilogramme which a man may make in twenty-four hours, according to Bouchard) is represented by the cipher 0.064. But the urotoxic coefficient of patients affected with dyspnœa of heart disease is always less. It oscillates between 0.0273 and 0.0370. Thus the diminished toxicity of the urine of myocarditis seems to be demonstrated and with it the fact that poisons are only incompletely eliminated by the kidney or destroyed by the liver. Hence follows the

supersaturation or intoxication of the blood announced by the clinical symptoms, dyspnœa, vertigo, delirium, and convulsions. In the beginning an exclusive milk diet may relieve the condition, but toward the end when the kidneys become impermeable and the liver insufficient, the dyspnœa becomes irremediable (Bouchard).

The attacks of difficult breathing show themselves more especially after effort, and constitute the paroxysms of so-called cardiac asthma. During the attack the expression is anxious, the face is pale and is sometimes covered with sweat. There is a feeling of imminent danger, but there is not the excruciating radiating pain which distinguishes attacks of true stenocardia. The patient finally succumbs in some such attack with the signs of suffocation from pulmonary œdema, or with syncope from heart failure.

Stasis in the lungs is announced also by cough, sometimes in bad cases by the expectoration of blood, though hæmoptysis is much more frequent in the hemorrhagic infarctions which occur in endocarditis. The signs of stasis in the extremities show themselves nearly always at the close of any exhausting disease in œdematous swelling of the feet and legs. But the dropsy of heart disease may be studied best in connection with endocarditis.

The incompetent heart soon suffers dilatation. The condition may be recognized on physical examination by the dislocation of the apex to the left and by the increase of dulness, which may extend to the right border of the sternum and beyond it. The dilatation may be so great as to produce relative insufficency of the mitral valve, which reveals itself in a systolic murmur at the apex, and in accentuation of the pulmonary valve sound. The condition is known to be relative by the fact that it disappears, to leave no trace. The fact is that endocarditis with valve lesions almost never occurs in diphtheria, scarlet fever, or typhoid fever, and the signs of mitral insufficiency in these affections usually disappear in the course of a few months.

The closure of the orifices of the heart is very much more complicated than has hitherto been believed. Both at the venous and arterial orifices the opening is narrowed by the contraction of muscle, and it is only by this contraction that the opening may be closed. The studies of Krehl have shown that the mitral orifice is reduced during systole to a narrow slit by the musculature which surrounds it. If this contraction does not ensue, the valves do not close the orifice. Parenchymatous degenerations are found at the base as well as in the papillary muscles. Interstitial processes do not so much affect the papillary muscles, but they do affect the musculature at the base.

The clinical signs of mitral insufficiency without the pre-existence

of acute rheumatism are, therefore, at least suspicious of muscle failure, and should lead to more careful examination.

In certain cases the dilatation of the right ventricle is so extreme as to lead to relative insufficiency of the tricuspid valve, which is recognized by the systolic murmur at the ensiform cartilage and by the venous pulse in the neck.

Forms.—The disease process is usually insidious and the symptoms are often latent or slight at first. Sometimes the symptoms are trivial for a long time, while the disease process gradually advances. The majority of cases of sudden death from unsuspected heart disease belong to this category. Usually, however, the disease is unsuspected because the condition of the heart has not been properly examined.

In another class of cases the symptoms seem to be out of proportion to the gravity of the lesions. These are the cases more especially of paroxysmal attacks of dyspnœa or syncope which disappear to leave the action of the heart in apparently normal condition.

In a third class of cases death is sudden, though never sudden in the sense that the arrest of the heart was absolutely unpreceded by signs of failure. Absolutely sudden death in the midst of apparent health, is very rare and even in diphtheria there are symptoms of some duration. Thus there is a comparatively latent period of two to ten days after the local signs, followed by weakness, apathy, somnolence in the day, insomnia at night, irregular and unequal pulse, hurried respiration, nausea, paroxysms of pain in the epigastrium, oliguria, albuminuria, finally, retardation of pulse, syncope, and death (Veronese).

Diagnosis.

The diagnosis rests upon the discovery of a demonstrable cause, that is, upon the history of some previous infection, especially typhoid fever, diphtheria, scarlet fever, pyæmia, etc.; or the myocarditis develops directly as the consequence of pericarditis or endocarditis in connection with the known causes of these affections.

The definite symptoms are diminution in the strength of the heart, the duplication, the so-called galop rhythm, muffling of the first sound, and sometimes the murmur of relative insufficiency which may be heard over the whole of the left ventricle.

If now to these symptoms there be added sensations of distress, uneasiness or pain in the region of the heart, dyspnœa, at first upon exertion, later more continuously, the character of the affection is better established. But the diagnosis is only absolutely assured with

the recognition of dilatation, which makes itself manifest with increase in the diameters of the heart along with the signs of loss of force. Increase in the diameters with increase of force indicates hypertrophy, but increase in the diameters with diminished force as shown by the lessened apex stroke, which is often dislocated downward and to the left, by the weakened sounds, and especially by the feeble pulse, indicates dilatation. Increase in the volume of the heart with diminution in the volume of the pulse is the surest sign of weakness of the heart muscle.

Beginning dilatation is indicated, further, by the signs of stasis, by the general duskiness of the surface, by ectasia of the small veins, especially in the face, and by puffiness of the subcutaneous tissue, which is noticed soonest in the back where the impress of folds in the garments or bedding remains a long time. This degree of dilatation is seen early, even in the first days of scarlet fever, but more frequently, though later in the history of the disease, usually about the middle of the second week, in the course of typhoid fever.

The diagnosis may be further corroborated by the diminution in the quantity of urine, by the signs of cerebral anæmia, by the evidence of stasis in the liver, marked especially by increase in volume, and often by attacks of so-called biliousness, and by icterus when the prime cause is a septic disease. Embolic processes in the lungs and kidney may throw additional light upon the case.

The diagnosis of the gouty heart is based upon the following points: 1. The presence of distinct gout. 2. The signs of latent gout, enterorrhagia, headache with nausea, irritable bladder, eczema, insomnia, and attacks of irritability and depression. 3. The alleviation of these symptoms by proper treatment, purgatives, gymnastics. 4. The presence of gout in the ancestry, with migraine, gravel, glycosuria, asthma (Bruce).

Suppurative processes may be suspected in the presence of chills followed by sweats, and when the myocarditis occurs in consequence of some septic disease, puerperal fever or other form of septicopyæmia. The discharge of pus by rupture into the pericardium produces the symptoms of a sudden intense pericarditis. Rupture of an aneurysmal dilatation of the heart into the pericardium is generally followed by sudden or rapid death.

Whether the diseased process is chiefly parenchymatous or interstitial it is impossible to say. Cases of sudden heart failure or extreme heart weakness show both sets of changes. So both interstitial and parenchymatous changes are found, as a rule, in diphtheria, while in typhoid fever the parenchymatous change, and in scarlet fever the interstitial change has been found most frequent.

Processes of slower development, as in subacute or chronic myocarditis, are diagnosticated by the abnormalities of the pulse, especially the various forms of arhythmia, with the more gradual supervention of the signs of weakness just described. The symptoms are typically represented in the senile heart, which shows signs of weakness and disturbed action upon any sudden movement, especially on arising suddenly from the recumbent or sitting posture. Later there are attacks of vertigo, confusion of ideas, and syncope.

Prognosis.

The prognosis must be guarded in all cases. Myocarditis is never a trivial affection; nevertheless most cases recover. The myocarditis of typhoid fever has, as a rule, a more favorable prognosis than that of scarlet fever or diphtheria, but heart failure is the most common cause of death in protracted cases of typhoid fever and in the sudden collapse of diphtheria. So long as dilatation is not pronounced the prognosis may be regarded as favorable, and though the heart may remain weak a long time in the convalescence of typhoid fever, it becomes restored by rest and recovers its natural tone in the course of a few weeks or months. As a test of the condition of the heart muscle Jaquet especially recommends the climbing of stairs, as the number of stairs climbed may more exactly gauge the gravity or extent of the degeneration. This author has constructed an "ergostat" which provides for the examination at once.

The myocarditis which occurs in connection with or as a sequel to pericarditis and endocarditis has a graver outlook. Bright's disease makes a bad prognosis. Purulent myocarditis is fatal. The suppurative process need not depend upon a grave septic inflammation, such as is seen in puerperal fever, sepsis, or pyæmia from any cause, as typical lesions have been described in connection with fatal infection by the gonococcus. Much depends also upon the character of previous treatment. When digitalis fails to secure relief, the outlook, as a rule, is bad. The prognosis as to entire restoration of the heart muscle must be guarded. The disappearance of symptoms does not always indicate a *restitutio ad integrum*.

Prophylaxis.

The prophylaxis consists chiefly in rest, and rest can be perfectly secured only in the recumbent or at least in the semi-recumbent posture. Every practitioner may recall cases of sudden death from heart failure in the course of diphtheria, typhoid fever, etc., from sudden violent or premature exercise of the body. The mere act of

arising in bed, of changing garments, of straining at stool has been followed by sudden heart failure, and that, too, in cases which could have been tided over the period of weakness by the observation of proper rest. So long as the pulse is feeble, small, or irregular the patient must observe rest, and the physician in no way better demonstrates his knowledge of the disease and his proper regard for the patient than by turning a deaf ear to all entreaty to be allowed to sit up in bed or to get out of bed too soon. This insistence will seem trivial only to him who has had no experience in these things.

Prophylaxis is further secured by the avoidance of the infections, by the use of antiseptics, by the abbreviation of the infections themselves, as by the use of antitoxin in diphtheria, by the saturation of the blood with the salicylates in rheumatism, by the deep injection method in the treatment of gonorrhœa, etc., etc.

Prophylaxis must have reference also to the avoidance of excess in gymnastics and in the various sports, of excess in the use of alcohol, which with any tendency to heart disease should be abjured altogether, of excesses in venery which always exhaust the heart and often prove fatal, of excesses at table, which act injuriously by increasing the blood pressure, and finally to the prevention of obesity.

Treatment.

The treatment of acute myocarditis requires absolute rest, without which is nothing. The slightest effort on the part of the patient may bring about a fatal collapse in an already exhausted heart. Muscular activity also increases the quantity of toxins which act injuriously upon the heart muscle. Under absolute rest the circulation may be secured without the action of the heart, or at least with only such action as will secure the opening and shutting of valves to permit the ingress and egress of blood under the influence of arterial pressure. What is the heart anyhow but a quadruplication of the aorta? The patient should, therefore, desist from every effort, and all motion of the body should be, as far as possible, purely passive. Thus the child affected with diphtheria may be easily handled by the attendant. The patient with typhoid fever, the old man affected with pneumonia, may be rolled over to the side of the bed to have the bedding changed, or may be lifted upon a sheet by a man at each corner, to be let down into a bath. Food and drink may be tendered in the recumbent posture; drink especially by the various devices for this purpose. The bladder and bowels must be evacuated with the proper vessels in bed. The physician will make his examinations by turning the patient over without lifting him in bed.

The value of rest in securing oxygenation of the blood was experimentally shown by Katzenstein, who found that "man requires during moderate muscular activity, from three to four times, and during violent muscular exertion from five to six times the quantity of oxygen needed in repose." And so with rest and the lapse of sufficient time the majority of cases of acute myocarditis recover.

An exclusive milk diet will itself sometimes cause œdema and dropsy to disappear. The writer has repeatedly seen patients gain in weight steadily under a half a gallon of milk a day, without other food than a light cracker or a piece of well-browned toast three or four times a day. Milk flushes toxins through the kidneys. Vichy water may be taken with it freely to obviate constipation. The food, other than milk, should consist chiefly of fruit and fish and the white meat of fowl. In a general way meat is better food for the heart than vegetables, but a mixed diet is best. The supper should be very light; let a glass of warm water, or at most a cup of very weak tea, suffice. Any imprudence in diet is quickly punished, any overfeeding is always hurtful. With rest and the regulation of diet there may be little need of more active medication. Thus Liebermeister reports of 234 cases of myopathic heart weakness without valve lesion treated at Tübingen in twenty years (1870–1890), 81 treated in this way without the exhibition of any drugs; and of these cases 46 recovered completely, 17 temporarily, and 18 only required further treatment.

In the treatment of chronic myocarditis it must be appreciated that the heart muscle may be rightly toned only by fresh air and exercise. It was a valuable suggestion and quite in accord with common sense that the heart muscle may be toned like any other muscle in the body. The effort to spare the heart had been already carried to extremes and degenerative processes favored by inactivity. It had been enjoined upon patients that all exercise was injurious. The importance of this injunction cannot be overestimated in acute myocarditis, where it is a question of sparing the heart until poisons may be eliminated from the body. But in chronic myocarditis other conditions prevail. The poison has been eliminated. The damage has been done and the heart muscle is left in a state of beginning degeneration. This process can be stopped in individual muscle fibres at the point which it has reached, and unaffected fibres may be stimulated to compensatory overgrowth by proper exercise. But the exercise must be graded to the individual case. Damage may be done by incautious methods. Thus climbing stairs is always hurtful, but climbing hills in the open air is helpful if practised aright. Fresh air feeds and exercise tones the muscle of the heart.

Much can be accomplished sometimes by change of climate, especially by sojourn at suitable watering-places. As a general rule, altitude is injurious, at least at first, as the heart may be over-stimulated in rarefied air. In bad cases the sea shore is best, or a stay upon the coast during the summer season. The rigors of winter may be avoided in our country by a sojourn in some of the Southern States, Alabama, Georgia, Florida, or the additional benefits of pure sea air may be secured in some of the adjoining islands, the Bermudas, the Bahamas (Nassau). Lighter cases or cases which have shown some improvement may get more benefit further north. Mountain resorts with shade and sunshine where the elevation is not too great, the Catskills, Adirondacks, the pine forests of the Carolinas, may tempt the patient to invigorate the heart with gradually increasing exercise in the open air. The exercise should never go beyond the point of dyspnœa or fatigue. It is profitable as well as pleasant in climbing to have stopping places on the route, at points where some prospect opens out. Lower California affords within limited regions every kind of climate. The Virginia Springs furnish every facility, including natural hot baths, for the proper treatment of heart disease. The baths of Nauheim have the highest reputation in Germany.

Patients affected with emphysema, asthma, and chronic bronchitis may learn to spare the heart extra effort, avoiding at the same time that extreme inactivity which conduces to degeneration and atrophy from disuse.

When the heart muscle is poisoned, especially by the toxins of any one of the infections, and the action of the heart is feeble, as indicated by the small, irregular, and arhythmic pulse, there is no heart tonic better than quinine, which is indicated whether there is or is no fever—but all the more if there is. To be effective it should be administered in small dose, as small doses (three grains every three or four hours) excite the heart, increase the strength of the pulse, and raise the blood pressure. As the remedy has no influence whatever upon the vagus, the excitement of the heart is ascribed to a direct action upon the heart muscle. Thus when it is a question of reducing fever in the face of a weak heart, quinine is to be preferred to any other antipyretic. Quinine has distinct and direct antimycotic properties. It destroys infusoria in a dilution of 1 to 20,000. It is supposed to exercise this influence by robbing the protoplasm of the power to take up oxygen. It stops fermentation and arrests decomposition, while it leaves the processes of digestion unaffected.

For steady support of the heart no remedy equals strychnine. Small doses of strychnine heighten the reflex excitability of the

spinal cord, so that the whole body becomes more sensitive to slighter impressions and the so-called vegetative functions are discharged with increased activity. It is really remarkable what improvement takes place at times in the general condition of old people under the use of a solution of strychnine or of the tincture of nux vomica. The appetite is increased; the bowels and bladder are more regularly and readily evacuated; the movements of the body are quickened; sleep is sounder and more restorative. The increased force of the heart is directly shown in the strength and volume of the pulse. Thus the brain is better fed, the eye brightens, interest in outside affairs is renewed, and new zest is added to life. Other forms of myocarditis besides the senile heart are influenced in the same way. But to be effective the dose should be small and gently stimulating rather than exciting. Thus the dose of the tincture of nux vomica may be from gtt. x. to xx. three times a day. Sometimes strychnine itself acts better. The preferable form is the nitrate in the grain to the ounce solution, of which the beginning dose may be five drops, to be gradually increased to ten drops three times a day. The effect is almost doubed if the remedy be given by subcutaneous injection. For this purpose, that it may not suffer change, the solution in the same strength should be prepared with one-half of one per cent. carbolic acid, and the dose may range from five to ten drops, that is, from the $\frac{1}{95}$ to the $\frac{1}{47}$ of a grain of strychnine, twice a day.

Heart failures of higher degree may be counteracted by the use of stronger heart stimulants. Digitalis stands at the head of these stimulants and is without a peer in the realm of materia medica. Digitalis contains a number of active principles, some of which are soluble in water, some in alcohol; hence choice may be had as between the infusion and tincture, or the virtues of all the principles may be secured by the use of the powder itself. The tincture is administered in the dose of five, ten, or fifteen drops in a teaspoonful or dessertspoonful of water every four to six hours; the infusion is given in the dose of a teaspoonful or dessertspoonful or tablespoonful at the same intervals; the powder in a dose of one to three grains. Digitalis acts slowly at first. It does not show its full effect until after the lapse of ten or twelve hours, and then the effect is continued or sustained for this length of time after the use of it is stopped. This effect is manifest in the increase in the force, and often in a corresponding reduction in the frequency of the pulse. It is seen also in the better blood supply to the brain and particularly in the increase in the secretion of the urine; as the quantity increases the color becomes lighter and more normal. Digitalis thus directly

tones the heart and eliminates toxins through the kidneys; hence the rapid improvement which shows itself under its use. Digitalis acts better in heart weakness from myocarditis than in valvular disease. It produces quicker and more permanent effects in myocarditis, and many cases recover absolutely under the judicious use of it. Thus Liebermeister reports of 172 cases of pure myocarditis, permanent recovery in 106, temporary recovery in 37, negative results in 29. When the heart's action becomes strong and the pulse is full and regular, it is wise to discontinue the use of the remedy, as too long-continued or excessive use may over-stimulate the heart and even bring about in it a condition of tetanus. The remedy, therefore, should be used for several days or for the greater part of a week, and the dose should then be reduced or better the remedy discontinued altogether, until such time as the condition of the heart and circulation may call for the renewed use of it.

Unfortunately, digitalis in certain cases disagrees with the digestion. It produces heartburn, distressing sensations in the region of the stomach, sometimes acid eructations, nausea, dyspepsia. In these cases it is necessary to substitute some other cardiac stimulant for the digitalis.

A good substitute for digitalis is strophanthus, which is best given in the form of the tincture. Strophanthus, like digitalis, in small doses increases the contractility of muscle and in fatal dose produces tetanic rigidity. Strophanthus also, like digitalis, has a special effect upon the muscle of the heart. In small doses it increases the force and lessens the frequency of the heart beats; it increases also blood pressure and with it the secretion of urine from the kidney. Strophanthus has an advantage over digitalis in that it acts quickly. The sphygmograph in the course of five to ten minutes shows the increase in the energy of the heart, which reaches its maximum in the course of one-half to one hour, and continues its effects for twenty-four to twenty-eight hours. Unfortunately, authorities differ as to the effects upon the blood-vessels. It was claimed at first as a great advantage (Frazer, Popper) that strophanthus, unlike digitalis, had no direct effect upon the blood-vessel walls. But Sée and Gley both observed an irritant influence upon the vasomotor centre and upon the ganglia of the vessels themselves. Still later investigations by Pawinski leave this point in dispute and seem to demonstrate a diminution in the tone of the vessels. At any rate, the effect must be much less than that of digitalis. The diuretic effect is observed usually only after the lapse of several days. It is seen sooner, often in the course of three days, in the presence of œdema, which may rapidly subside under its use. As individuals

differ in susceptibility, it is best to commence with a small dose, three to five drops, and gradually increase it to ten to fifteen.

The active principle, strophanthin, is sometimes recommended for subcutaneous use. It is, however, little suited for this purpose on account of the intense local irritation which may ensue, pain, redness, infiltration, sometimes suppuration.

A better substitute for subcutaneous use is found in sparteine, which proves of especial value in cases marked by arhythmia, palpitation, pressure, precordial anxiety, and the predominance of nervous symptoms, hysteria, neurasthenia, Basedow's disease, etc. Sparteine is given in the dose of one-fourth of a grain several times a day. Larger doses, even up to one grain, are sometimes administered, but the effect is better with small doses and the remedy may be administered in this way continuously for weeks.

Thus, to invoke the aid of mnemonics, the tincture of strophanthus is given in about the same dose as that of digitalis, and the sulphate of sparteine in about the same dose as the sulphate of morphine.

For quick effects, as to bridge over an impending collapse, a powerful remedy is found in camphor, which may be injected subcutaneously dissolved in ether 1 to 10, in syringeful doses every fifteen or twenty minutes. A valuable adjunct is furnished in caffeine, which in the form of the sodium benzoate may be administered internally or subcutaneously; internally in the dose of five grains or subcutaneously in the dose of three grains in the same way.

It will be remembered that the heart may be roused to activity by appeals to the skin, as by friction over the whole surface or that of the extremities, as by dry mustard, by hot applications, bottles of hot water along the spine and at the feet. The stimulating effect of a big sinapism over the whole præcordium is not to be despised.

In the development of the heart muscle it will be remembered also that the centres for respiration, vaso-motor action, and cardiac stimulation lie close together in the medulla, and that stimulation of any of these centres is communicated to the others. The action of the hot bath in dilating the surface capillaries or relieving the heart of tension is seen at once in the reddening of the surface of the whole body. The warm or hot bath is indicated when the heart is weak and dilated and is unable to overcome the blood pressure at the periphery. The hot bath, therefore, acts best in relief of the sense of fulness associated with pain and dyspnœa, in acute myocarditis, or in acute exacerbations of the chronic form. The action of cold baths is demonstrated in the glow which follows the first contraction of the capillaries of the surface. So the use of the daily cold bath is a powerful means of invigorating the heart in chronic myocarditis.

ENDOCARDITIS.

Acute Simple and Septic Endocarditis.

Endocarditis (ἔνδον, within); inflammation (infection) of the endocardium.

History.

Endocarditis was first recognized as a distinct and separate disease by Bouillaud, who published an account of it in his treatise on the heart and vessels in 1824. Individual reports of affection of the lining membrane of the heart had been made in the last century by Boerhaave, Morgagni, and Sénac, and in the early part of our century by Wells, Baillie, and Kreysig. Kreysig, in fact, attributed disease of the valves to inflammation of the endocardium and recognized this affection as a frequent complication in the course of scarlet fever and rheumatism. But it was reserved for Bouillaud to distinctly connect rheumatism with this affection and to individualize the inflammation with the definite name endocarditis. Corvisart had already recognized the *frémissement cataire* as a symptom of disease of the mitral valves, and Laennec had made it possible to differentiate in life every form of valvular lesion. But neither of these acute observers had apprehended the character of endocarditis or appreciated the anatomical foundation of the lesion. By the aid of the signs furnished by Laennec, Bouillaud was able to diagnosticate the existence of endocarditis and localize it with great accuracy upon some particular valve. Bouillaud also called attention to the fact that other diseases than rheumatism might produce the disease, pointing to pneumonia as a not infrequent cause. He appreciated the fact further that the acute form was often attended with signs of pyæmia, and thus, in the recognition of the etiological relation of rheumatism and pneumonia, in the picture of pyæmia presented by the acute form, and in the anatomical localization of the disease, the foundation of the whole modern pathology, Bouillaud became the pioneer in the development of the doctrine of endocarditis.

The next period in the history of endocarditis belongs to Virchow, who, with his simple description of the process of embolism, explained the hitherto obscure complications on the part of the brain, lungs, and kidneys. There was as yet, however, no satisfactory account of the exact nature of the deposits upon the valves, that is, of the thrombi which went to form the various emboli. Virchow, himself (1856), was inclined at first to regard the granular deposits them-

selves as microscopic parasites. It could not be known at that time that these deposits were the products of parasites, and the discovery of this fact constitutes the third period in the history of the disease.

The pioneer in these investigations is Klebs (1878), who attributed all forms of the disease to micro-organisms, and insisted upon it that the various bacteria of any one of the different infections might colonize on the endocardium and produce endocarditis. Next followed Köster, with his more precise demonstrations. Individual observations had been made by Winge, of Christiania (1869), and by Heiberg (1872) in which the presence of bacteria in the vegetations had been distinctly remarked. The distinction had already been made by Fraentzel of simple or benign and ulcerative or malignant forms. Klebs showed that both these forms resulted from the action of bacteria. A little later all support for any etiological division between these forms was withdrawn by the demonstrations of Litten, who found the same micro-organisms in both forms and showed that there was no abrupt, but only a gradual difference between the two forms.

The new era began with the findings of Weichselbaum, Wyssokowitsch, Fraenkel, and Sänger (1885). These observers turned out a number of micro-organisms, especially the pyogenic micro-organisms, the presence of whi h was established also by culture and inoculation. About this time Banti found in twenty cases the streptococcus seven times, the staphylococcus once, both these micro-organisms three times, the diplococcus lanceolatus eight times—the findings in two cases being negative. Next followed the demonstrations of Netter and Weichselbaum (1886) of the diplococcus of pneumonia in the products of ulcerative endocarditis in the course of pneumonia, and in the same year the disclosure by Heller of tubercle bacilli in the endocardium. Thus the view originally expressed by Klebs, that endocarditis might occur in the course of any one of the infections, has gradually been established by demonstrations. The latest contributions go to show that the bacterium coli and the gonococcus may also produce the disease.

Endocarditis being produced by so many causes, is a quite common affection. It is difficult to estimate its frequency with any exactness, as statistics are usually based upon chronic endocarditis. It is known, however, that certain cases of acute endocarditis terminate as such, although most cases affect the valves of the heart. Where the lesion is confined to the walls of the heart it may produce no symptoms and thus be overlooked. The frequency of chronic endocarditis is variously estimated at from one to two per cent. (Rosenstein), five per cent. (Willigk), eleven per cent. (Foerster), seventeen per cent. (Chambers).

Etiology.

It may be considered now as an established fact that endocarditis results from the colonization upon the endocardium of the micro-organisms of an infectious disease. Endocarditis is, therefore, never primary; it is always a secondary process. As all the blood in the body must pass through the heart continuously, it is not surprising to learn that any one of the pathogenic micro-organisms may excite inflammation in the endocardium. The question of interest concerns only the frequency of infection by the different diseases of this character and the rôle of any predisposing cause, as trauma, exposure to cold, or previous disease of the heart or its vessels, as in the processes of atheroma and arteriosclerosis.

To study the subject first from a clinical standpoint. The connection with rheumatism has been remarked from the start and the predominating influence of this affection in the production of endocarditis has always been recognized. It is said that from sixty to eighty-five per cent. of the cases of endocarditis are caused by rheumatism. As already stated, Kreysig called attention to this fact first, and Bouillaud derived nearly all cases from this disease. Bouillaud declared, in fact, that "in any acute articular rheumatism which was at all severe or extensive, the coincidence of a pericarditis or an endocarditis is the rule, the law, and non-existence is an exception." This relationship is now so well established as to be known by the people.

But the frequency of this affection is differently estimated by different authors. Budd, Fuller, Latham, found in 425 cases of rheumatism 154 cases of endocarditis and 124 of pericarditis. Sibson found in 325 cases of rheumatism more or less distinct endocarditis 130 times, endo-pericarditis 54 times, and pericarditis alone 9 times. Wunderlich estimated the proportion of cases of endocarditis in rheumatism at $\frac{1}{10}$ per cent., Jaccoud, Ormerod, Bamberger, and Duchek at $\frac{1}{4}$ per cent., Sibson at $\frac{1}{2}$ per cent. If every case of irregular or tumultuous action of the heart is to be considered as an endocarditis, the highest of these ratios is not enough, and if only those cases are to be so regarded in which permanent lesions are left, the lowest might suffice. It is certainly true that the heart may be disturbed in its action without the development of endocarditis, and it is equally true that many cases of endocarditis recover without permanent lesions. *In medio tutissimus ibis*, the truth lies between these extremes. It is now conceded that about one-third of the cases of acute articular rheumatism are followed by endocarditis.

In all cases it must be remembered that rheumatism is a mantle which covers many affections. Many forms of rheumatism are followed by endocarditis in only exceptional cases. Further, it must be remembered that septicæmia, which is the cause of many cases of ulcerative endocarditis, is not real rheumatism, or is something more than rheumatism. Reference is had in this consideration only to acute articular rheumatism. This rheumatism certainly furnishes the greatest contingent of cases of endocarditis, and it is especially the more severe forms with extensive implication of the joints that are liable to this complication. Endocarditis is rather the rule in multiple, but is the exception in monarticular, chronic, muscular, and gonorrhœal affections. The situation of the joint affected has little or no influence. Endocarditis is as liable to supervene after affection of the joints of the lower as of those of the upper extremity or trunk.

Of all the factors, the most important is that of the age of the patient. It is the young who are especially liable to this complication. But rheumatism itself is not a disease of infancy. Most of the cases so reported are cases of septicæmia, syphilis, etc. Clinicians with the widest opportunity for observation record but isolated cases of real rheumatism in the earlier weeks of life. But when rheumatism does occur in infancy it is especially liable to be followed by endocarditis. West, for instance, declares that the proportion of cases is 61.5 per cent., Picot estimates it at 78 per cent., Cadet de Gassicourt finds it as high as 81 per cent. As endocarditis is most frequently caused by rheumatism, the disease is most often found in the third and fourth decades of life. An original outbreak of endocarditis is rare in advanced life and most of the cases at maturity are survivals from youth and adolescence.

The period of development of endocarditis varies much. In cases of multiple joint affection in young people, endocarditis is wont to set in early, often in the course of the first week. In less grave infection and less numerous involvement of the joints, endocarditis may not occur until after the lapse of ten days or two weeks. Sibson found that most of his cases occurred in the course of the first week, Lebert his in the course of the second week. Potain declared that two-thirds of the cases occurred before the tenth day, but that in some cases the complication did not develop until the seventeenth, twenty-second, or even the thirty-seventh day.

Sometimes these delays refer to the period of recognition. The physician who makes a habit of carefully examining the heart upon every visit soonest recognizes the development of endocarditis. The diagnosis is often made only after the existence of the disease for

some time. Eloquent in this regard is the experience of Potain, who diagnosticated endocarditis for the first time in twenty-two of thirty-five cases upon entry into the hospital.

It remains to be remarked that as in the case of pericarditis, the endocarditis may be the first serous membrane attacked, so that occasionally inflammation of the endocardium precedes the affection of the joints. Observations to this effect were made long ago by Bouillaud, Graves, and Trousseau, and more recently by Fernet, Martineau, and Halez. The interval between the endocarditis and the joint affection may vary from two or three up to twelve days (Hache), often up to fifteen days (Jaccoud). The fact that endocarditis may precede the rheumatism, and that either affection may exist independently of the other, has developed the belief that neither one of these affections causes the other, but that both are effects of a common cause.

What is the cause of rheumatism?

Klebs claimed to have found the first cause in certain monadines, to which he ascribed pathogenic relations to both affections. Cornil and Babes refused to accept this view, and a number of subsequent observers, Hamberger, Fraenkel, and Weichselbaum, failed to find these micro-organisms in the vegetations of endocarditis. Lion attributes the failure to the fact that investigations are made at too late a period in the history of the disease. Petit appeals to the feeble vitality of certain micro-organisms in explanation of the fugacious localizations of rheumatism. This author claimed to be able to find a parasite of remarkably short duration of life in acute articular rheumatism. It is certain that blood taken in the period of fever, pain, and swelling of the joints, that is, during the height of the disease, is always sterile (Wurtz).

It is believed at the present day that acute rheumatism is caused by micro-organisms, probably closely allied to but not identical with the micro-organisms of pus. Goldscheider demonstrated bacteria in the serous exudation of pleurisy, which he believed to be due to rheumatism. Upon cultivation these micro-organisms turned out to be staphylococci. Sahli (1892) developed cultures of a micro-organism from the exudation of endocarditis, which turned out to be the Staphylococcus citreus. Leyden (1894) found a diplococcus which differed from the staphylococcus in that it did not grow upon the usual culture soils but would grow only in human blood serum. These micro-organisms were delicate, round micrococci, which differed as much from the diplococcus of pneumonia as from the streptococcus of pus. Subcutaneous injection of these micro-organisms produced moderate but never severe fever and no local reaction.

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Howard found a micro-organism in the exudation of ulcerative endocarditis which was in every way identical with the bacillus of diphtheria. Gourmont and Le Clerc cultivated a micro-organism taken from the blood of a woman suffering with ulcerative endocarditis a few hours before death. They developed a diplococcus which did not liquefy gelatin and whose individual elements were larger than those of the staphylococcus. It did not form itself into chains or masses.

Experimental studies have been successful in producing endocarditis with so many micro-organisms as to have led to no definite results. Jousserand and Roux succeeded in producing a severe endocarditis in the rabbit by the inoculation of a coccus which was cultivated from blood taken in life from a woman suffering with endocarditis, affected also with swelling of the spleen, high fever, and epistaxis. This coccus was a staphylococcus of coarser granulation than the Staphylococcus aureus. It was like it in the fact that it did not liquefy gelatin.

Thus it may be seen that nothing definite is as yet known regarding the specific micro-organism which must be the cause of rheumatism. It may be said of acute articular rheumatism, as was long ago said of croupous pneumonia, it is such a distinct affection that it must be produced by a specific cause, the disclosure of which, probably in the near future, will enable the clinician, as in the case of pneumonia, to draw the lines more definitely about doubtful cases.

The avenue of entrance may be by the throat. Buzz especially noticed the frequent connection between angina and acute rheumatism. In twelve families the two affections repeatedly occurred together. Further, this observer reported fourteen cases in which an attack of acute rheumatism followed directly in the course of an angina. Many similar cases may be found in literature in which the cause of the angina is stated to be the pathogenic staphylococci and streptococci. These cocci of angina have but little virulence and hence do not produce suppuration in the joints.

In this connection may be discussed next chorea and erythema nodosum, affections which are closely allied to rheumatism. Chorea was connected with heart disease as long ago as in the time of Bright (1833), but the relation of chorea to rheumatism is by no means clear. We are concerned here only with the connection between chorea and endocarditis. Statistics vary in this regard. Lebert, Strümpell, and Eichhorst found the connection exceptional. Roger claimed to have found endocarditis alone 47 times in 71 cases of chorea and 19 times in connection with pericarditis. Prior found the coincidence only 5 times in 92 cases of chorea. Schott found valve diseases nine times

in chorea. Rosenbach saw one case in which the endocarditis preceded the chorea for several days. The attempt has been made to explain chorea as the result of embolic processes in the brain. It is known, however, that chorea occurs at times in the course of rheumatism without the intervention of endocarditis, and, as Rosenbach shows, embolic processes have been demonstrated in only a very limited number of cases. The fact that chorea occurs so frequently in the course of pregnancy, and terminates with the pregnancy, points to the circulation in the blood of some toxin which affects the nervous system. So chorea has been derived from a toxæmia which produces all three affections, rheumatism, endocarditis, and chorea. They may coexist or either of these affections may exist alone. Endocarditis is certainly a frequent occurrence in severe cases of chorea, and Pepper finds that few fatal cases of chorea fail to show some warty excrescences on the valves.

Erythema nodosum has been seen in connection with endocarditis by Trousseau, Gubler, and Roger, and by numerous later observers. It may be said of this erythema, as has just been remarked of chorea, that it may coexist with rheumatism and endocarditis, one or both, and each one of these infections may exist alone. It is probable, therefore, that the same cause produces all three conditions.

After rheumatism the next most important rôle in the production of endocarditis is played by the so-called septic diseases. Simpson as long ago as 1884 noticed the occurrence of endocarditis in the puerperium, and a host of subsequent observers, commencing with Virchow and concluding with the classic investigations of Litten, have studied the development of endocarditis in the course of the puerperal fevers. It has long been known that a peculiar gravity was imparted to puerperal fever by the occurrence of endocarditis, which with its associate myocarditis often gives the *coup de grace*. But it is now known that every form of endocarditis, the simple verrucose as well as the grave ulcerative and diphtheritic varieties, may occur in the course of any case of septicæmia. The old ideas of the production of a disease under a rheumatic dyscrasia have been gradually displaced by the demonstrations of the micro-organisms of septicæmia.

The so-called surgical sepsis falls under the same head, and endocarditis has been frequently observed in cases of osteomyelitis, periostitis, erysipelas, furunculosis, dysentery, and other affections caused or attended by the micro-organisms of pyæmia and septicæmia.

The Streptococcus pyogenes is most frequently found in the cases of endocarditis which occur in the course of puerperal fever. Here

the uterine mucosa forms of course the avenue of entrance. The Staphylococcus pyogenes aureus has been most frequently found in connection with furunculosis, anthrax, suppurative wounds of the skin, osteomyelitis, bronchiectasis, etc. From the various infarctions and miliary abscesses which occur in the course of these diseases, pure cultures of this staphylococcus have been developed. The avenue of entrance of these micro-organisms is often evident, as in the case of puerperal fever or affections of the skin, erysipelas, furunculosis, etc. Sometimes it may be discovered when search is made for it, as in a case of more or less secreted caries of bone, periostitis, affection of the prostate gland, etc. In other cases the cause remains concealed. Such cases are said to be cryptogenetic. We owe our knowledge of cryptogenetic septicæmia especially to Leube. Jürgensen afterwards demonstrated the influence of concealed sepsis in bacteriological studies. Sometimes the cause in these cases is revealed upon autopsy. Thus Lewa reported two cases of endocarditis, in one of which the infection seemed to have been derived from the bile ducts, which were found dilated and contained concretions, but no pus. In the other case there was an abscess the size of an apple in the pancreas filled with fetid pus; there was also suppurative meningitis. Both of these processes were considered secondary. In Mason's cases, women aged eighteen and twenty-seven, the affection apparently originated in Fallopian tubes filled with pus. In the case of Ingraham and Wolcott the infection originated probably in the puerperal uterus. Paulus demonstrated two cases of septic endocarditis which had developed on the basis of old processes of the mitral valve. This observation showed that sometimes the septic infection may remain a long time in the body without producing general sepsis, but that certain definite but unknown causes may produce new local and general disease. In this case the author believed that the death was caused by the invasion of the staphylococcus which had entered eleven years before. During the invasion the patient suffered repeated attacks of rheumatic polyarthritis. The final attack was caused not by a rheumatic but a septic virus, which led to a general septic infection.

Endocarditis in the course of erysipelas was first noted by Gubler and afterward was especially studied by Jaccoud and Sulzer. Gendram reported a case of septic endocarditis consecutive to a traumatic erysipelas of the face, and Balassa reported a case of endopericarditis in the course of erysipelas of the leg. The streptococcus has been demonstrated in these cases, but whether as a primary or secondary invasion—that is, whether as the streptococcus of erysipelas or of pus—is not accurately determined, as the difference between

these micro-organisms has not yet been clearly established. The streptococcus of erysipelas has been isolated in endocarditis, and from this streptococcus endocarditis has been experimentally produced in animals by Valliard and Vincent, Vidal, and Besançon.

In this connection may be studied also the endocarditis which occurs in the course of gonorrhœa. The first case of gonorrhœal endocarditis in which the cause was accurately demonstrated was reported by Leyden in connection with myocarditis. Contrary to common expectation, the literature shows a number of cases in which this connection could have been established. The first case was reported by Brandes, in 1854. Subsequent cases were reported by Hervieux, Voelker, Lehman, and Brouardel. Some of the cases recovered and some terminated fatally. His reported two fatal cases. Hitherto these endocarditic deposits have been attributed to the streptococcus and staphylococcus, and have been considered as mixed or secondary infections, but in the case reported the gonococcus of Neisser was found in the polypous deposit. The gonococci had the distinct biscuit form and lay for the most part in characteristic manner inside of cells. They could be colored by the method of Gram and decolorized by alcohol and lavender oil. As blood serum is such a peculiarly favorable soil for the development of the gonococcus, the colonization of it on the endocardium is not surprising. The demonstration of it in this region is of great value in explanation of gonorrhœal affections of other serous membranes. Endocarditis has been noticed often in connection with the rheumatism of gonorrhœa, but also occurs entirely independent of the joint affection. Thus Lion reported from the literature six cases of endocarditis in twenty-two cases of gonorrhœa without rheumatism. The rheumatism and endocarditis may occur together or independently, because they are both produced by the same cause. Councilman, Wilms, and Michaelis corroborated these disclosures in subsequent reports.

The endocarditis which occurs in the exanthemata may be due to pathogenic micro-organisms whose nature has not yet been established, or may be due to secondary invasions by the micro-organisms of pus. The very first observers noticed endocarditis in the course of scarlatina. Thus Bouillaud, Graves, Trousseau, and Roger all reported cases. West saw endocarditis three times in thirty-nine cases of scarlatina. The complication may develop itself at any time in the history of the disease, during the eruption, during desquamation, or during the existence of joint affection. Litten and Bokai encountered a number of micro-organisms in the endocarditis of scarlatina. Henoch and Fraenkel actually demonstrated a streptococcus in the deposits of endocarditis.

Variola is rarely followed by endocarditis, though cases have been reported by the earlier observers, as Bouillaud, and more recently by Huchard and Brouardel. Moulinier found a number of cases in the course of the epidemic of 1870. As might have been expected, endocarditis occurs more frequently in the severe cases of confluent variola, and the intensity of the poison is indicated by the fact, noticed by Brouardel, that the endocarditis is not usually isolated, but is generally accompanied by pericarditis and myocarditis. In the case recorded by Huchard, endocarditis developed as early as the third day, though usually it occurs later, in the course of the second week of the disease.

Still more infrequently does endocarditis occur in the course of measles, though cases here too have been noticed by Bouillaud, Stokes, and Roger. Wunderlich seemed to have found the complication frequent, but the experience of most subsequent observers coincides with that of Parrot, who found not a single case of endocarditis in 800 cases of measles.

Endocarditis is very rare in the course of diphtheria. Statements of the frequency of this complication date from a period when the nature of diphtheria was not so accurately established. Thus Labadie-Lagrave reported the occurrence of endocarditis in twenty-two of forty cases of diphtheria. Parrot called in question the accuracy of these observations. In fact, Howard reports the single case in which the Klebs-Löffler bacillus, or a bacillus which answered to all the descriptions of the diphtheria bacillus, was demonstrated in the products of the valves.

Typhoid fever is also an infection which is rarely followed by endocarditis, though individual cases have been recorded by Bouillaud, Skoda, Hoffmann, Guéneau de Mussy (Petit). These cases will not all bear strict analysis, as in many instances the diagnosis was based upon a bruit, which may occur in myocarditis. It is well known that the toxin of typhoid fever expends itself chiefly upon the myocardium, but the Koch-Ebert bacillus of typhoid fever was actually demonstrated upon the valves in a case of endocarditis by Jeriot, and endocarditis was produced experimentally in the rabbit by the intravenous injection by Lion of the typhoid bacillus. The rarity of endocarditis in the course of typhoid fever coincides with the well-established fact that the typhoid bacillus is rarely found in the blood. Some of the few cases which occur can be accounted for as coccus invasions. Thus Klebs found a micrococcus in the verrucose masses; Fraenkel and Sänger demonstrated the Micrococcus albus and aureus, Sänger also the streptococcus in the vegetations. In some of the cases the Bacterium coli has been incriminated as the

cause of the condition. Étienne found the Bacterium coli on the surface of the vegetations six hours after death in a case of ulcerative vegetative endocarditis.

After rheumatism, pneumonia is the most frequent single cause of endocarditis. In fact, the pneumonic endocarditis is perhaps the best established and best known form of the disease, having been directly demonstrated, as stated, by Netter and Weichselbaum in 1886. Bouillaud and Legroux had already noticed the development of endocarditis in the course of pneumonia and regarded the complication as frequent. Niemeyer, on the other hand, thought it exceptional, but modern observers, Klebs, Netter, and Weichselbaum, have demonstrated its relative frequency. Demonstrations are now made absolute bacteriologically. Thus Venturi reported a case of endocarditis with cerebro-spinal meningitis in the course of croupous pneumonia. The heart was found of normal size. The aortic valves were affected with circumscribed ulcerative endocarditis. Histological examination of the valve tissue showed suppuration of the endothelium with fibrinous deposits, containing leucocytes, red blood corpuscles, and diplococci in great number.

Saintsbury also reported a case of ulcerative endocarditis and meningitis after pneumonia. Typhoid symptoms set in, and in this connection a double soft murmur at the base of the heart established the diagnosis of malignant endocarditis with meningitis. Section revealed on one of the valves of the aorta a soft vegetation the size of a hazelnut, into which protruded a small aneurysm. A microscopic examination of the vegetation revealed short bacilli with bipolar coloring. The diplococcus of pneumonia localizes itself upon the valves of the heart, especially in cases of old lesions which have been perhaps hitherto unnoticed. It may occur in any part of the course of pneumonia, most frequently, however, at or after the period of crisis, which it sometimes interrupts, but occasionally as late as a week after the subsidence of fever.

Moreover, as in the case of pericarditis, the diplococcus of pneumonia may localize itself upon the valves of the heart in the entire absence of any affection of the lungs. Thus Barbacci described a case of purulent pericarditis with meningitis and endocarditis in a child aged seven years. In this case the microscopic examination revealed the exclusive presence of the diplococcus of Fraenkel. The finding was verified by culture. There was no pneumonia. Thus endocarditis has occurred in the course of cerebro-spinal meningitis, which is produced by the same cause as pneumonia, and the diplococcus has been found in the products of valve lesions in the entire absence of any sign of disease elsewhere. The aortic valves are most

frequently affected in these cases, and especially in the ulcerative form. Netter made a number of observations of this kind. This observer also produced endocarditis experimentally in the rabbit by engrafting the pneumococcus after traumatism. It is questionable if the pneumococcus may produce the lesion upon perfectly sound valves. In man it is usually some old lesion which acts as the predisposing cause. The rôle of the pneumococcus has been especially studied by Klemperer, who found the characteristic diplococcus in the exudation of endocarditis, cultivated it, and demonstrated its virulence by the inoculation of rabbits.

Catarrhal pneumonia may be followed by endocarditis, but here the lesion is always secondary, as catarrhal pneumonia itself is a secondary process. The streptococcus is the cause of the endocarditis in these cases, but as croupous and catarrhal pneumonia sometimes coexist, it is not surprising to learn that the diplococcus and streptococcus have been both discovered in the lesions of endocarditis in the course of these diseases.

Influenza is produced by a bacillus of its own, but endocarditis has been observed in the course of influenza by Pawinski and by Oulmont, Barbier, and Huchard. Naldoni reported a case of endocarditis which announced itself with dyspnœa, palpitation, and pain four days after an attack of influenza. A systolic murmur could be recognized at the apex. The diagnosis of acute endocarditis was established. The patient recovered but the diagnosis was confirmed two months later on autopsy by the finding of insufficiency and stenosis of the mitral valve. In all these cases, however, the streptococcus was the micro-organism discovered, so that the endocarditis was the result of a secondary infection.

Tuberculous endocarditis was observed long ago by Corvisart, and has since been described by Potain. Kidd claims to have found it twenty-seven times in 500 cases. Hanot declared that he could not discover the tubercle bacillus in the deposits on the valves and attributed the disease to toxins produced by other micro-organisms. But Cornil, Babes, Kundrat, and Rindfleisch demonstrated the bacillus in the superficial layers of the granulations. Tuberculous endocarditis has been found hitherto exclusively in cases of acute tuberculosis. The tuberculous matter occurs as small, hard, caseous particles of yellow color, or as granules to constitute vegetations. Courmont showed to the Society of Medical Sciences at Lyons a guinea-pig which he had inoculated with the deposits of an acute endocarditis in a tuberculous patient. The guinea-pig became tuberculous. Courmont and Dor found that when attenuated tubercle bacilli were injected into the blood of rabbits the emaciation only

occurred after the lapse of half a year, and then set in tuberculous inflammation of the joints which corresponded to the tumor albus of man. In the citation of this connection it was observed that the cause of the disease was sometimes the streptococcus and sometimes the staphylococcus, sometimes again the Friedländer capsule bacillus or the Fraenkel pneumococcus.

Syphilis expends its poison chiefly upon the vessels. It does not usually attack the heart, and then rather the myocardium. But occasional cases of syphilitic endocarditis have been reported. Engel-Reimers reported two cases of syphilitic endocarditis. In one case death occurred with cerebral signs. Endocarditis was diagnosticated by the action of the heart and other symptoms. Section revealed verrucose excrescences on the mitral valves with a thrombus on the ventricular surface of the anterior valve. There were infarctions in the spleen and kidneys and emboli in various arteries. A second case showed a papillary syphilide. After several chills a murmur was heard at the heart. Death occurred in collapse. The section revealed verrucose deposits on the mitral valve and a thrombotic mass as large as a cherry. There were infarctions in the spleen and kidney. This case had been diagnosticated as typhoid fever, but there was no sign of affection of the intestine.

Greene also reported a case of syphilitic endocarditis. The patient, a young girl, showed a secondary syphilitic exanthem, with systolic murmur at the base of the heart. Syphilitic endocarditis usually results from extension of the disease from the myocardium.

Moore reported a case of endocarditis with miliary abscesses of the heart in a case of hip-joint disease, Stintzing a case of endo- and pericarditis in the course of typhoid fever, and Lécorché saw fourteen cases in the course of diabetes. The origin of the affection in these cases is usually very insidious.

Bright's disease much more frequently causes pericarditis than endocarditis. The endocarditis which develops in the course of Bright's disease may be an extension of the inflammation from the pericardium, or it may result as a secondary affection from some of the other complications which show themselves in the course of Bright's disease, rheumatism, pneumonia, etc. Diabetes and gout act in the production of pericarditis rather as predisposing causes. Diabetes especially, by lowering the bactericidal properties of the blood, favors the development of pathogenic micro-organisms and the action of toxins. Gout has something of the same effect, with more direct action upon the blood-vessels in the processes of arteriosclerosis.

Endocarditis may occur in the course of malaria. Griesinger

spoke of this connection. Duroziez saw grave valvular lesions in twenty cases of old malarial affection, and Lancereaux went so far as to localize the preference of malarial affection at the aortic orifice. The modern view is that of Kelsch and Kiener, who are unwilling to admit the direct influence of malaria in these cases, ascribing the endocarditis rather to secondary infection (Petit). Malaria is a term so much abused that it should be discarded altogether.

The causes of endocarditis are by no means exhausted in this array of the infections. It is universally admitted that even the most trivial infection may be followed by endocarditis. Thus endocarditis has been observed after affections as inconsiderable as quinsy and mumps. Bouchard and Karth reported unmistakable cases of endocarditis in the course of parotitis. Lannois and Lemoine went so far as to attribute the cerebral accidents which sometimes occur in the course of mumps to emboli of cardiac origin.

The tendency of modern belief is to regard the throat as the avenue of entrance for the micro-organisms of rheumatism, and endocarditis has been reported in the course of both simple and epidemic tonsillitis (quinsy). Buss believes it probable that acute rheumatism is produced not by a single but by a number of micro-organisms, and that rheumatism, like pneumonia, acute osteomyelitis, and endocarditis may be caused by various attenuated pathogenic micro-organisms. Czerny reported a case of endocarditis following ritual circumcision. The case was that of a child aged four weeks. The wound healed slowly but was otherwise normal. Fever set in with murmurs at the heart. Shortly thereafter there was increased dulness in all directions. The child was dismissed in these conditions.

In the vast majority of cases endocarditis is produced by micrococci. Thus Banti found in 66 cases collected from the literature, in which positive results were obtained by bacteriological study, one species of bacterium alone 47 times, several species associated 19 times. Of the 47 cases the Staphylococcus pyogenes aureus was found 13 times, the Staphylococcus albus once, the Streptococcus pyogenes 8 times, and the Diplococcus lanceolatus 15 times.

In the 66 positive findings the Streptococcus or Staphylococcus pyogenes or the Diplococcus lanceolatus was demonstrated 51 times. In the other 15 cases there were found various micro-organisms.

It would appear as if the Diplococcus lanceolatus had preference for the aortic valves and the pyogenic cocci for the mitral valve. And it would appear as if the diplococcus, when it was found, was rather more frequently found alone, while the others are oftener associated.

Besides these well-known species other observers have encoun-

tered other micro-organisms of different character. Sometimes these micro-organisms have not yet been found in other affections. Thus Lion reported the finding in one case of a bacillus much like the Bacterium coli, but more immobile and with highly toxic filtered cultures. This bacillus produces in the rabbit a vegetative endocarditis, often without any previous lesion of the valves. Weichselbaum and Netter described a particular bacillus of endocarditis as a short, mobile, micro-organism which resembled the typhoid bacillus in its culture upon gelatin and the Bacillus coli in its culture upon potato. Weichselbaum produced with this micro-organism an experimental endocarditis after a lesion of the aortic valves. This same observer also found a particular micrococcus in a single case of endocarditis affecting both valves, and succeeded in seven experiments in producing endocarditis in the dog after lesion of the valves. This micro-organism had no effect upon the rabbit. Weichselbaum also found a capsulated bacillus something like the bacillus of Friedländer. It was found in pairs, in fours, or in six or eight bodies in the same capsule. He succeeded in producing endocarditis with this bacillus. Endocarditis may be produced also by the Bacillus fœtidus of Fraenkel and Sänger. This micro-organism was found in two cases, once alone, once in association with the Staphylococcus pyogenes. This bacillus produced endocarditis after injury of the valve, attended with small ulcerations, on the surface of which the bacillus could be detected. On another occasion Weichselbaum found a non-cultivatable bacillus, once isolated and once associated with the pneumococcus and streptococcus. Many of these micro-organisms are considered to be varieties of the Bacterium coli. Wurtz thinks that the case of Netter and Martha of suppurative endocarditis supervening in the course of biliary abscesses was probably produced by the Bacterium coli.

Whether an endocarditis may ever occur in man under the direct action of micro-organisms or their products, or whether in all cases some previous lesion of the valves is necessary, is a question still unsettled. It is certainly true that the majority of cases show the evidence of pre-existing lesion, usually of atheromatous, cicatricial, or sclerotic character.

Klemperer and Levy also emphasize this point with the statement that in all diseases and also in malaria and carcinoma the concomitant endocarditis does not need to be an effect of the original infecting pathogenic germ, but that it may be the expression of a secondary or mixed infection which attaches itself to the endocardium weakened by the primary infection. We may find then in the deposits the staphylococcus, the streptococcus, or the diplococcus, in

short, the micro-organisms which we have learned to know repeatedly as the cause of secondary infections. It is also a question if in these cases the disease may not be produced by some toxin, that is, by some chemical irritant. Gerhardt and Wagner support this view. But that the lesion may be produced by the direct deposits of the cause has been proved, so far as cancer is concerned, in one case by Kundrat, who found cancerous nodules as a secondary deposit upon the surface of the valves.

It has been seen that but few micro-organisms are sufficiently virulent or erosive to affect the valves without the intervention of previous lesion. These lesions need be only very slight, such, for instance, as might occur under severe strain, anything that would lead to an abrasion or desquamation of layers of the endothelium. Rokitansky believed that the preference of the right heart in fœtal life was due to the prevalence of defects which invited the affection on this side. The extent of the lesions may also have much to do with the virulence of the affection. At least it is known that the extent or severity of the endocarditis does not necessarily correspond to the character of the originating disease. As already stated, mild cases of rheumatism may, in exceptional cases, produce malignant endocarditis, or grave cases of pyæmia may produce a mild endocarditis. Much will depend here upon the virulence and number of the micro-organisms. After a lesion of the valves any of the various micro-organisms of infection or of suppuration introduced by endovenous injection or by catheterization of the right carotid artery have been found to produce an endocarditis of malignant character. The same result may be reached without this severe interference if the material which contains the bacteria be introduced in larger particles, for instance, particles from a potato culture. Deviti found bacilli in vegetations in a case of heart disease in the course of typhoid fever, which he considered as the typhoid bacilli. In other cases of endocarditis this observer found the diplococcus, staphylococcus, Bacillus fœtidus, and was able to produce endocarditis in animals by the injection of these micro-organisms into the serous cavities. In his experiments the valves were previously injured or the matter was mixed with sterilized meal to make it stick better.

The so-called malignant or ulcerative endocarditis may be, etiologically and clinically, nothing more than the usual endocarditis with necrosis of the vegetations. In some of these cases the micro-organisms have been demonstrated circulating in the blood of the living patient (Klemperer and Levy).

Nevertheless grave cases of endocarditis, that is, cases of the so-called ulcerative, diphtheritic, or malignant form of the disease, are

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usually produced by the pyogenic micro-organisms. It has even been proposed to remove these cases from the category of heart disease altogether and consider them under the head of pyæmia. Litten showed that an infectious process in any organ, in the uterus, for instance in the puerperium, may show all the symptoms of ulcerative endocarditis without the manifestation of the slightest lesion of the endocardium. Leube calls up in this connection the cases of spontaneous or cryptogenetic septicæmia, in which the primary avenue of entrance is unknown and in which the manifest secondary localizations are considered the direct cause of the disease. Rosenbach puts it right when he says that the chief danger in these cases is not the valve lesions but the sepsis, of which the valve lesion is only one of many localizations. Petit quotes from many authors as the avenue of invasion of micro-organisms which have either primarily or secondarily produced endocarditis: the skin in accidental or surgical wounds, suppurations, furunculosis, panaritium, ulceration of the upper lip, gangrene; the bones, osteomyelitis, vertebral caries, otitis media; the mucous membranes, chiefly from the genital tract, and most frequently from the uterus after labor, after abortion, affections of the prostate, of the urethra, of the bladder, of the kidneys, of the seminal vesicles; the digestive tract, gangrenous stomatitis, gastric or intestinal ulcer, dysentery, typhoid fever, tuberculosis, etc.; the bile passages; the respiratory passages, suppurative affections of the lungs, bronchiectasis, etc.

As stated elsewhere, valve lesions in intra-uterine life are almost confined to the right side of the heart, and in extra-uterine life to the left side of the heart. As valve lesions are mainly produced by endocarditis, this preference applies to the localization of the disease. Statistics from the Berlin Pathological Department show the lesion in 300 cases, on the left side in 297, on the right side in 32; confined to the left side alone 268, and to the right side alone but 3. The very reverse conditions prevail in the fœtus and the new-born child. Rauchfuss found fœtal endocarditis located in the right side 192 times, and in the left side but 15 times. Mackenzie found endocarditis of the right heart but twice in 38 cases, and in each of these two cases the left side of the heart was attacked in slight degree.

Endocarditis affects by preference the mitral valve, next in frequency the aortic valves; in the common endocarditis of pyæmic origin the mitral valve is always affected, and in 51 cases of rheumatic endocarditis Barié found mitral lesions alone 40 times, mitral and aortic lesions 3 times. The tricuspid and pulmonary valves are attacked in only the most exceptional cases, and then, as a rule, consecutively to repeated attacks of the valves of the left side of the heart.

Sex makes but little difference. Valve lesions from endocarditis are rather more frequent in the female sex. Bamberger maintains that aortic insufficiency is more common in men, and mitral stenosis is more common in women. Marshall found in 508 cases of mitral stenosis 350 in the female, and 108 in the male sex. Endocarditis is rare in infancy, but begins to show itself with the occurrence of the various infections.

Endocarditis may be divided, according to the intensity of the symptoms, into acute and chronic forms, and according to the character and duration of the lesions into simple, septic, and sclerotic forms.

Conclusions.—The etiology of endocarditis may be epitomized as follows:

(1) Endocarditis is always a secondary disease, the result of the action of some infectious process.

(2) The infections which most frequently cause endocarditis are, in the order of their frequency, rheumatism, pneumonia, septico-pyæmia, tuberculosis. But any infection, however trivial, may suffice to introduce a cause of the disease. Endocarditis is the least frequent in the course of typhoid fever and diphtheria, diseases which attack by preference the myocardium. It is only fair to state here, however, that the experience of different observers is not uniform in this regard, and most at variance is that of Schott who found, in 62 cases, endocarditis produced by scarlet fever 19 times, typhoid fever 16, influenza 8, diphtheria 7, measles 7, intermittent fever 3, and puerperal fever 2 times.

(3) Rheumatism and pneumonia more frequently produce mild and benign, while septico-pyæmic affections more frequently produce grave or malignant cases. There is, however, nothing in the nature of the infectious process which necessarily determines the character of the lesion in the valves; rheumatism may produce grave affection and sepsis may produce light affection.

(4) In certain cases the specific micro-organisms of definite diseases directly attack the endocardium, in other cases the endocarditis is a secondary infection.

(5) Sometimes the micro-organisms, whether primary or secondary, act directly upon the endocardium; in other cases the action is produced indirectly under the deleterious influence of toxins.

(6) Most of the cases of endocarditis in the course of rheumatism may be regarded as a localization of the rheumatic poison in the heart; cases of grave or malignant endocarditis produced by pyogenic micro-organisms should be regarded as local manifestations of pyæmia or septico-pyæmia.

Morbid Anatomy.

Endocarditis is the result of the direct deposit of micro-organisms upon the surface of the endocardium and the penetration of bacteria to the depths of the tissue. The multiplication of these micro-organisms or the influence of the toxins which they develop leads to a more or less extensive necrosis with destruction of tissue, the cells of which lose their nuclei and become converted into granular matter. The affected region shows, therefore, the appearance of a thrombotic mass without cellular elements. Upon the rough surface thus formed fibrin is detached from the blood to present the exuberant verrucose or cauliflower appearance. Where particles of fibrin are aggregated to form masses of considerable size, they constitute what are called the polypoid growths, and when under the process of necrosis the superficial layers are sloughed off or mechanically detached, the condition is known as the ulcerative or diphtheritic endocarditis. In this process of necrosis and destruction of tissue the wall of the endocardium is sometimes so much thinned as to yield to the pressure of blood and constitute aneurysm of the heart, especially of the valves, and where the thin wall is finally broken through, the valve becomes perforated so as to prevent perfect closure and permit regurgitation. Valvular aneurysm is found only in the left side of the heart. These aneurysms are usually of globular form or more or less cylindrical, and show an opening which in the case of the mitral valve is turned toward the cavity of the ventricle and in the aortic valves is opposite the side of the vessel, that is, at the point of greatest pressure under closure of the valves. They are usually very small, the size of a pin head to a pea, but may be as large as a nut or even as a small egg. In such case the aneurysm is composed of the whole of the valve distended to an extreme degree (Petit). The wall of the aneurysm consists of fibrous tissue and the cavity contains coagula which are sometimes stratified or even more or less organized. Soft clots may be the source of emboli. Sometimes in this process of destruction a valve is partially or entirely separated from its attachments.

Whether the process of endocarditis as it affects the valves results from infection of the free surface by the blood which circulates in the chambers of the heart, or is carried to the valve in blood-vessels which penetrate between the layers, is a question still in dispute. Coën claims that the semilunar valves are entirely free of vessels, while the auriculo-ventricular valves are provided with a thick network some branches of which extend to the points of inser-

tion of the chordæ tendineæ. Moreover, fine vessels extend from the papillary muscles deep into the chordæ tendineæ. The micro-organisms colonize usually deep in the substance of the valve and are separated from the true tissue by a layer of cells free of nuclei. Beyond this necrotic zone is a layer of cells rich in nuclei, as if armed against intruders.

In the first stages of the infectious process the delicate endothelium loses its lustre, becomes opaque, and under the exudation of serum, swollen and more or less weighted down. The process may stop at this stage, the exudation may be absorbed, the opacity may disappear, and the valves may be restored to a perfectly normal condition. Even destructive lesions may be repaired under the processes of hyperplasia. Particles of fibrin, cauliflower excrescences, or polypoid growths may be detached and swept off into the distant circulation to constitute emboli. In this way the normal function may be restored to previously incompetent valves, and anomalous sounds which have indicated the existence of this condition, may entirely disappear. In other cases the thrombotic mass undergoes subsequent degenerative change especially into hyaline, fatty, or chalky matter, and losses of tissue are repaired by proliferation of the connective tissue which makes itself manifest as cicatrices in the structure of the endocardium.

The granular fibrinous matter is deposited along the line of closure of the valves at the point of contact at some distance from the free margin of the mitral and tricuspid valves, and closer to the edge of the semilunar valves, in festoons which have their point of contact with the free margin at the nodules of Arantius. The deposits are usually of grayish or light reddish color, are tough and elastic as they are formed by hyperplasia of the superficial layer of the valve tissue, by connective tissue, and by superimposed fibrin. Sibson called attention to the fact that vegetations in the mitral valve are usually deposited on the auricular surface at a distance of 2 to 3 mm. from the free edge, while on the aortic valve they are deposited by preference on the ventricular surface along the line of the lunated spaces. In both cases the deposits occur at the surfaces of maximum contact.

Köster attributes the localization of deposit at the line of closure to the fact that the vessels which enter the valves break up into a fine plexus and form a network along this line. The explanation would hold good at least in the case of the auriculo-ventricular valves. Darrier found in healthy new-born children vessels in the valves only in the case of the venous valves, but in pathological cases vessels were found in all the diseased valves. Rosenbach remarks that anomalous

valves, especially in the aorta and pulmonary artery, always predispose to endocarditis, and, as already remarked, in normal valves the slightest lesions of any kind act as predisposing causes.

The fact that the mitral valve is most frequently affected lends support to the view that in the majority of cases micro-organisms or their products reach the valves through the nutritive blood-vessels and do the first mischief from within. After the surface of the valve has become roughened and disturbances of nutrition have led to desquamation, further changes are produced by deposits from the blood circulating in the heart. The blood-vessels which supply the valves are found in a state of inflammation. They show distinctly the processes of endarteritis, which in the course of time, through thrombotic occlusion, develops into endarteritis obliterans. This fact explains the rapidity with which sclerotic changes occur in the unaffected valves. The process of infection carries with it its own destruction in the obliteration of the nutritive vessels.

These sclerotic changes vary in extent in every degree. Sometimes the process is limited to a slight thickening or opacity of the valves, which may be scarcely distinguished from the senile or friction sclerosis. In other cases one or more valves become so thickened as to be absolutely incompetent, or the sclerotic change may bind together contiguous valves and thus by occluding the lumen of the orifice offer an obstacle to the egress of blood. The occlusion of an orifice is favored by the fact that the process of thickening is attended by shrinkage of the valve, so that the thickened tissue is protruded as a rigid mass into the calibre of the tube. Sometimes the valves are bound down to the wall of the heart or of the issuing vessels so as to effectually prevent closure and permit regurgitation. In other cases the process expends itself more particularly upon the endocardium which covers the tendinous cords, or the papillary muscles, and thus binds down or interferes with the free play of the valves. Where destructive changes set in at the same time, the tendinous cords may be torn in two or separated. This action occurs with especial frequency in the development of the atheromatous ulcer.

In all cases of any duration the disease process extends to involve the myocardium. The nature of the original disease may have much to do with the associate myocarditis, but the duration of the endocarditis is the most essential factor.

The condition of the heart in valve disease was thoroughly studied by Krehl in eight hearts, six of which were affected with mitral insufficiency and stenosis, two with aortic insufficiency and mitral stenosis. The muscle of the heart was examined microscopically layer by layer. In this extremely thorough study the musculature

was found to show very marked changes in all cases. There was pericarditis usually, always endocarditis, changes of the small vessels, thickening of the intima, multiplication of the nuclei, hyaline degeneration, partial sclerosis of the greater arteries, proliferation of the connective tissue, partly in the neighborhood of the vessels and the peri- and endocardium, partly in depots and masses, sometimes in round-cell infiltrations. The musculature was destroyed in the connective-tissue mass. Fatty degeneration of the muscle cells existed in only slight degree. Perforation of the septum occurs usually at the part free of muscular tissue in the "undefended" region of Peacock under molecular necrosis at this site (Reinhardt, Hanschka).

Valve disease, therefore, entails progressive inflammation of the heart, which injures it in high degree and naturally affects the prognosis, far more, in fact, than the valve lesion itself.

The hypertrophy which ensues with this consecutive change or as a result of interference with the circulation has already been sufficiently discussed. Further changes in the valves themselves remain to be described under the subject of valve lesions.

The extent of the lesions in the heart and the character of the complications are illustrated in the following cases collected from the literature:

Peacock reported a case of endocarditis with destruction of one of the aortic valves and partial perforation of the septum of the ventricles, with fatty degeneration of the heart, and another case resulting in partial destruction of two of the aortic valves and perforation of the base of the mitral valve; Talamon a case of endocarditis of the septum with the development of inter-ventricular aneurysm; Pierot a case of acute endocarditis with aneurysm of the mitral valve which led to a mechanical insufficiency and sudden death; Lilley a case of septic endocarditis with suppurative atheroma, septicæmia and death; Barben-Dubourg a case of septic endocarditis with aneurysm of the heart, communication of the left ventricle and right auricle, without infarction; Cloquet a case of septic endocarditis with ulceration and perforation of the left auricle; Keating a case of septic endocarditis with pyæmia, in which death occurred from perforation of the heart; Caubet a fatal case of septic endocarditis with aneurysm of a sigmoid valve and of the inter-ventricular wall and myocarditis; Conplaud a case of septic endocarditis with vegetations on and perforations of the aortic valves with aneurysm of the mitral valve and splenic infarctions; Lépine a case of septic endocarditis with aneurysm of the sigmoid valves of the aorta and multiple emboli; Dianaux a case of acute endocarditis with valvular aneurysm, making communication between the aorta and the right ventricle; Peyrot a case of acute

endocarditis followed in three weeks by aneurysm of the inter-auricular septum and by the development of two secondary aneurysms protruding into each auricle; Ogle a case of ulceration, partly of the anterior curtain of the mitral valve and partly of the anterior and left portion of the wall of the left auricle, with the appearance of an aneurysmal pouch produced by adhesion of fibrin to the margin of the ulcerated opening; Malherbe a case of septic endocarditis with ampullary aneurysm of the aorta; Ekelund a case of endocarditis with simultaneous affection of the mitral valves, of the semilunar valves of the aorta, and the arch of the aorta (aortitis); Potain a case of septic endocarditis with the formation of a voluminous clot at the mitral orifice and with obliteration of the aorta at the bifurcation; Bianchetti a case of acute endocarditis affecting the mitral and tricuspid valves followed by dilatation of the left ventricle and right auricle and acute exudative pericarditis; this case recovered; Law reported a case of endocarditis affecting both mitral and tricuspid valves with ruptured chordæ tendineæ; Langer cases of septic endocarditis of the tricuspid and pulmonary valves, with embolism of the pulmonary artery; Rodet a case of septic endocarditis with almost complete obstruction of the mitral valve, dilatation of the left ventricle and of the pulmonary artery, with aneurysm of the inferior mesenteric artery; Millard a case of vegetating endocarditis with voluminous polypoid concretions which occluded the left auriculo-ventricular orifice; La Sayre a case of endocarditis with softening of the heart, latent peritonitis and death, and Pergami a case of perforating ulcer of the septum of the heart followed by sudden death.

Moxon reported a case of septic endocarditis with abscesses in the brain and spleen; Pepper a case of septic endocarditis affecting the aortic valves, with embolism of the kidneys, jaundice, and death in nine weeks; Charcot and Vulpian a case of acute septic endocarditis of typhoid type with ulceration of the tricuspid valve and formation of multiple abscesses in both lungs; Carrié a case of vegetative endocarditis with infarction of the spleen and kidneys and embolism of the right radial artery; Féréol a case of septic endocarditis attended by obstruction at the aortic orifice, caused by an aneurysm of the septum occurring in pneumonia, with apoplectic infarction and infarction of the spleen, and Curtis a case of septic endocarditis with thrombosis and extensive gangrene.

Symptoms.

Endocarditis usually develops insidiously. The condition is disclosed for the most part because it is expected and sought after, and not because of the presence of obtrusive symptoms. As already inti-

mated, endocarditis sometimes exists for years unrecognized and unsuspected. The reserve force of the heart suffices to overcome or to compensate the lesions; therefore the unsuspected conditions are more frequently found in earlier life where nutrition is good and where the muscular force is strong. Some intercurrent disease, more especially an infectious process, most frequently a renewed attack of rheumatism, makes manifest a lesion hitherto entirely latent. Sometimes the condition is called out by a sudden or excessive demand upon the heart beyond the reach of the reserve force.

Much depends also upon the situation of the lesion. Endocarditis which is confined to the walls of the heart seldom shows signs, while affection of the valves or of the parts concerned in the manipulation of the valves is more quickly manifested. But even in such cases the lesions may be so disposed as to interfere but little with the action of the heart, so that sometimes even extensive lesions may be latent. Caroli demonstrated such a case to the Royal Academy of Medicine, Dublin, in the heart of an apparently healthy young man who had died suddenly. The heart showed signs of a former malignant endocarditis. Death occurred in consequence of embolism in the coronary artery caused by a few particles from a thrombus. Small thrombi were found on the chordæ tendineæ of the anterior segment of the mitral valve. Nauwerck described mural endocarditisand its relation to the so-called spontaneous heart failure. Mackenzie reported a case of septic endocarditis which perforated the left ventricle but did not affect the valves, and Bellingham a case of latent endocarditis with large deposits of lymph on the valves of the heart. An almost incredible case of latent valve disease was recorded by Cobb. A woman, aged twenty-two, examined by a number of physicians, showed not the slightest anomaly on the part of the heart up to her death, when section revealed immense cauliflower vegetations at the mitral valve, which almost blocked the orifice, and also extensive vegetations on the aortic and tricuspid valves. For what length of time endocarditis may remain latent or show but trifling signs becomes apparent in the statement of Sir Andrew Clarke, who reported six hundred cases of heart disease which showed no serious symptom for more than five years.

On the other hand, the disease announces itself at times with distinct and even violent signs. The patient may be seized suddenly with severe pain in the region of the heart, with dyspnœa or cyanosis; the action of the heart itself may be greatly excited, show arhythmia or the galop rhythm, while the organs of the body may display the signs of defective or insufficient blood supply. Sometimes a sudden strain is the immediate cause of rupture of a valve whose

structure has been previously softened or changed by the processes of endocarditis or atheroma. Zunker reported a case of this kind, and Fraentzel another case in which excessive strain in extracting a harrow from soil which had become dry, was the direct cause of insufficiency of the aortic valves. Fraentzel records also the case of a physician who had reached advanced life, and who, having in a long and active practice attained a small competency in a small town, retired to Berlin to enjoy the peace of old age. But arteriosclerosis soon developed under the ease of his new situation. For the treatment of this condition he entered a private hospital, where he suffered no particular distress until one day he stooped suddenly from the desk on which he was writing to seize a piece of paper which the wind had blown away. In the moment of stooping he was seized with a violent pain in the region of the heart, and a severe attack of dyspnœa. On examination it was found there was an exquisite insufficiency of the aortic valves. Under a rest of three days and small doses of digitalis the symptoms subsided, but to return again in the course of a few weeks and this time to remain five days, up to the death of the patient. Autopsy revealed remarkably rigid valves without any trace of endocarditic deposit. One of the valves showed a quite recent scar running from the free margin of the valve to the base, reddish in color and covered with a light coagulum which could be easily detached. The posterior valve showed a distinct rupture running across its structure in a zigzag course from the free border at the middle of the valve to the base. Small recent coagula were deposited on each side of the line of rupture.

The fact is, however, that such cases are exceptional and the grave signs of disturbance of the heart, especially dyspnœa, cyanosis, and dropsy belong not so much to affection of the endocardium as of the substance of the heart itself.

Acute endocarditis usually announces itself by symptoms which are variable and inconstant, so that most cases escape recognition for a long time, and the diagnosis is finally awarded to the investigator who is most conversant with the distant as well as the immediate results of infection of the endocardium.

Fever is a sign which frequently leads to the recognition of endocarditis. There is, however, nothing characteristic in the temperature curve. The fever is irregular. It may be absent entirely. What gives it significance is the fact that it develops, or if previously present is increased, in the course of rheumatism without any evident cause—that is, there is no extension of inflammation in joints already involved or there is no implication of new joints. An elevation of temperature from two to three degrees excites the suspicion of

the practitioner, who proceeds to establish the existence of endocarditis by physical signs. If the fever shall have been preceded by chills, or if chills occur irregularly in the course of an infectious process, there is all the more reason to suspect endocarditis as one of the manifestations of septico-pyæmia. These chills may recur with such regularity as to simulate intermittent fever, and cases of endocarditis have been for a long time overlooked under this diagnosis. In other cases the chills are more irregular and erratic. These cases, too, are often, under superficial observation, dubbed malaria and dismissed, or are more properly interpreted as evidence of pyæmia, though the localization in the heart may be unrecognized. Irregular chills with sweats especially indicate septic endocarditis.

Sometimes the attention of the practitioner is directed to the heart by the alteration in the character of the pulse. A previously regular pulse becomes irregular, or a pulse which had simply shown the increase in frequency which belongs to any fever, now bounds under the excitement of the heart produced by irritation of the endocardium. Reference has been made elsewhere to the irregularity and excitement produced in the substance of the heart in a reflex way by irritation of either the pericardium or endocardium.

At the same time the patient complains of suffering from palpitation of the heart, which is evident in the tumultuous action and which causes a more or less active or vague distress. The palpitations set in especially after any exercise or emotional excitement or occur in attacks or paroxysms which may show themselves at any time during the day or even in the night. These attacks of palpitation are also to be interpreted as evidence of reflex irritation.

Dyspnœa is not, strictly speaking, a sign of endocarditis. The difficulty of breathing belongs to the interference with the action of the heart itself under reflex excitement or as the result of disease of the myocardium. Dyspnœa belongs rather to the cardiac asthma which occurs in the later stages of valve lesions, and is to be interpreted as a sign of heart failure rather than as evidence of endocarditis. Any positive pain present is due to neuralgic complication or to association with pericarditis.

Headache is a common symptom in the beginning of the disease; there is more or less insomnia; there is nearly always anorexia. The digestive apparatus is usually distinctively affected, but more especially in cases which assume the typhoid form. In these cases the tongue is thickly coated, especially in the middle, while the borders are often entirely clean. Along with sordes there is stridor labialis and nasalis. It requires constant efforts on the part of the attendants to keep the tongue moist. The patient expresses no wants but takes

drink with relish and avidity, especially cold water or soda water, less willingly milk.

The abdomen is not much distended. The bowels may act naturally, and either constipation or diarrhœa is exceptional. Diarrhœa, especially with discharge of bloody stools, or dysentery would point to embolism of the mesenteric arteries.

The spleen is always swollen. It is often tender to the touch. The enlargement may be recognized when the patient lies upon the back, but is often appreciated only when the posture is changed to the right side, when the weight of the spleen may carry it forward and downward by gravity. In a lighter case the patient may stand, and in this way an enlarged spleen is sometimes discovered. With patients in bed the spleen may usually be felt by palpation, when it is found to be hard and painful. Anything like a sensation of friction imparted to the hand would indicate hemorrhagic infarction. Swelling of the spleen is so uniform in acute endocarditis as to be absent only when enlargement is prevented by old perisplenitic processes (Fraenkel).

The liver is often somewhat enlarged and sensitive to pressure from the parenchymatous swelling which occurs in all the infectious diseases.

Palpation of the abdomen sometimes reveals tenderness over the kidneys. The urine is dark-colored, occasionally because of the presence of blood, and shows a moderate amount of albumin, blood-corpuscles more or less crenated under the action of the urine, and sometimes distinctive micro-organisms. These symptoms are clearly signs of toxæmia. It need not be repeated that all these symptoms vary in every degree of intensity and any one of them may be entirely absent in the beginning of the disease.

Physical Signs.

During the development and in the acute stages of the disease the physical signs may also be unobtrusive and unreliable. Inspection may reveal tumultuous action with palpitation, under which it may be seen that the impact extends over a greater area. In other cases the contrary condition is observed. The action of the heart is weakened so that even the apex stroke is no longer to be seen. These conditions may be more distinctly appreciated by palpation. The action may be so violent as to make manifest a vibratory thrill, or in other cases the fingers fail to find the situation of even the apex stroke. This weakness of the heart becomes especially marked in the later stages of any form of the disease.

Percussion furnishes at first only negative evidence. Later, in the

course of the disease it is seen that the outlines of the heart are increased. This increase in the diameters of the heart is the most valuable of all signs in the diagnosis of chronic endocarditis. It will be remembered that the heart swings in the cavity of the chest suspended by its great vessels, and is subject to some dislocation with the various movements of the body; but for practical purposes the apex is found in the fifth intercostal space, two inches below the nipple (in the male) and one-half an inch to the right of the left mammillary line. The base of the heart lies about the level of the upper margin of the third rib. The dulness of the right border begins at the third costal cartilage, extends downward along the left margin of the sternum, curving slightly to the right, to the end of the sternum. The dulness of the left border extends downward and outward in a curved line from the third costal cartilage to the point of the apex beat. The lower line of dulness runs from the apex to the end of the sternum. These outlines are not materially increased at the start, but in the course of time, with the development of hypertrophy, the diameters of the heart are increased in every direction. They may in extreme cases extend from the second rib to the diaphragm and from the right border of the sternum to the left mammillary line.

Auscultation reveals the most valuable signs in the very inception of the disease. Inasmuch as endocarditis affects chiefly or at least attacks first the mitral valve, disturbance in the action of this valve is made manifest by anomalies of sound or by murmurs. In the great majority of cases the mitral valve is so affected as to prevent perfect closure, so that upon contraction of the ventricle blood regurgitates from the left ventricle into the left auricle. The first sound of the heart, which is made chiefly by muscular contraction, but also to a considerable extent by closure of the mitral valve, is thus changed or substituted by a bruit which is appreciated in its greatest intensity at the apex and is synchronous with the systole of the heart. The murmur is usually, at first at least—because due at first to muscular paresis—of soft blowing character; so that a murmur of this character in this region, in connection with other signs of the disease, may establish the diagnosis. It will be understood that if the action of the heart be very weak the first sound or the murmurs which substitute it may be entirely absent. One of the signs of extreme heart weakness is failure of the first sound. It will be understood also that endocarditis affecting the columnæ carneæ or the chordæ tendineæ would have the same result. Much depends also upon the character of the lesion. Tough fibrinous deposits may produce louder and rougher sounds. Soft vegetations may not greatly interfere with the current of the blood and may exist to considerable extent at times

without the production of a murmur. Further, floating vegetations may be washed into the current of the circulation so that murmurs previously present may disappear. The mitral valve may be affected in such a way as to offer obstacle to the free ingress of blood from the left auricle. In this case any audible murmur would be presystolic. Affections of the aortic valve would give rise to murmurs heard in greatest intensity at the base and synchronous according to their character with the first and second sounds of the heart.

Interference with the circulation soon leads to compensatory hypertrophy, and under this hypertrophy certain valve sounds are distinctly intensified or accentuated. These accentuations are more distinctly audible in connection with the closure of the sigmoid valves of the pulmonary artery and the aorta. When dilatation occurs rapidly, as in cases of associate myocarditis or when the hypertrophy shall have given way in the further course of the disease, the valves, especially the tricuspid valve, fail to close the orifice, with a development of murmurs which indicate relative insufficiency. These various murmurs will be studied in detail in connection with chronic valvular disease.

Secondary Symptoms.

There remain now to be considered the distant effects of endocarditis. The interference with the action of the heart under the irritation produced by the infection of its lining membrane disturbs the blood supply to the various organs of the body. In the stage of excitement the blood pressure is increased, and this increase is especially noticed in the brain. There may be headache, insomnia, and flushing of the face. Any disturbance of the intellect at the start is to be attributed rather to the toxæmia of the originating malady than to the cerebral disturbance which occurs in rheumatism, especially in cases marked by hyperpyrexia. In the later course of the disease, or in grave (septic) affections in the earlier stages, the blood supply is deficient. There is, along with the weak action of the heart, anæmia of the brain, evidence of hyperæmia in the lungs, a disturbed respiration, stasis in the kidneys, manifest in deficient excretion of urine, and still later dyspnœa and dropsy, which begins at the feet and gradually mounts up the extremities. These conditions are, however, rarely observed in the acute stage of the disease; they belong to chronic endocarditis.

A much more distinct and characteristic picture is furnished by the processes of embolism which result from the detachment of thrombotic masses from the endocardium to be lodged in distant organs of the body. In this process of embolism the brain suffers frequently.

The detachment of a thrombus may be a very insidious process, and the lodgment of an embolus may provoke little or no disturbance when collateral circulation can be quickly established. Very different, however, is the result when the embolus lodges in terminal vessels where collateral circulation cannot be established. Such terminal arteries are found in parts of the brain, in the lung, spleen, and kidneys. The sudden occlusion of vessels of any magnitude in these organs is announced by immediate and often severe signs. Thus the process of embolization is usually marked by chill, sympathetic vomiting, fever, pain, hemorrhage, and arrest of function. Further symptoms will depend upon the organ affected and upon the character of the embolus. The merely mechanical emboli, plugs of fibrin, coagula of blood, plaques of atheroma, etc., may produce no other symptom than inhibition of function; the septic embolus is attended with destruction of tissue, with the formation of metastatic abscesses, which may in turn supply material for further destruction.

Embolism of the brain occurs most frequently in the branches of the left carotid artery, whose trunk lies more directly in the course of the circulation. The thrombotic mass detached from the heart finds its way through the carotid artery to branches of the artery of the fissure of Sylvius. Occlusion of a large branch of a brain artery produces an apoplexy with loss of consciousness, coma, and hemiplegia. On account of the situation of the embolus, aphasia is a common manifestation. Most of the cases of apoplexy which occur in youth or middle age result, not from cerebral hemorrhage, which is the common cause of the condition in advanced life, but from embolism of an artery of the brain in the course of endocarditis, which is itself in turn chiefly a consequence of rheumatism. So well understood is this fact that the practitioner always makes a careful examination of the heart in every case of apoplexy in earlier life. Uræmia, which produces the same symptoms at the same time, is usually easily excluded by the fact that in these cases the sudden stroke was preceded by a train of nervous signs. Sometimes the examination of the heart furnishes only negative evidence, because the very masses which would *in loco* have made themselves manifest by signs have been washed away to constitute the emboli.

Infected emboli develop metastatic abscesses in the brain, usually with meningitis. Westphal called attention to the psychoses with acute delirium which occur in the puerperium in the course of ulcerative endocarditis. Rosenbach insists upon it, however, that there was always in these cases an individual disposition which was the chief factor of the psychic alienation.

The abolition of consciousness may be so abrupt in cases of embolism of the brain as to prevent the appearance of other signs.

The causes, course, and complications of embolism of the cerebral arteries are exemplified in a few cases selected from the literature. Thus Prevost reported a case of septic endocarditis in the course of suppurative pneumonia, followed by softening of the island of Reil, from obliteration of the Sylvian artery, attended by a phlegmon of the neck and obliteration of the right jugular vein with right hemiplegia and aphasia; Darolles a case of septic endocarditis in connection with pneumonia at the apex in an alcoholic subject; aortic insufficiency developed rapidly and was followed by meningitis of the convexity; Sokolowski a case of acute endocarditis followed by embolism of the right Sylvian artery and softening of the brain; Stintzing a case of septic endocarditis in the course of a cryptogenetic septico-pyæmia, which proved fatal through embolism of the artery of the fossa of Sylvius; and Hayem reported a case of subacute septic endocarditis attended by splenic and cerebral emboli, multiple infarctions, softening of the brain, and death.

Embolism of the lungs presents a more typical picture of the general process. Lodgment of the plug in a vessel of any size is attended by severe pain, by sympathetic vomiting, and by dyspnœa, which becomes extreme in proportion to the size of the vessel occluded. Under the process of infarction which now develops, hemorrhage occurs with the expectoration of frothy mucus tinged with blood or of a larger quantity of fluid black blood. Occlusion of one of the chief branches of the pulmonary artery is attended by extreme anxiety, by cyanosis, convulsions, suffocation, and syncope. A certain number of cases of sudden death in endocarditis result from occlusion of a main branch of the pulmonary artery and not from heart failure.

Embolism of the liver shows itself with chills, pain in the region of the liver, considerable swelling with tenderness and especially with icterus. It will be remembered that icterus may occur independent of any direct implication of the liver or catarrh of the bile ducts. Icterus shows itself in pyæmia as a result of decomposition of the hæmoglobin. In these cases there is no true jaundice as the fæces remain colored; in fact, there is often polycholia from increased activity of the liver.

Embolism of the spleen shows itself also in a chill, fever, and severe pain in the region of the spleen, which is evidently enlarged and is tender to the touch.

Embolism of the kidneys presents a more characteristic picture. After the chill, vomiting, and pain in the region of the kidneys, the

evidence of occlusion is furnished in the disturbed nutrition of the capillaries, which permit the exudation of albumin and blood. Albuminuria and hæmaturia are, therefore, characteristic signs of infarction of the kidney.

Embolism of the mesenteric arteries is revealed by colicky pain in the abdomen, and diarrhœa, with the discharge sometimes of fetid black blood. As the mesenteric arteries are not strictly terminal, the initiatory signs, chill, vomiting, and fever, may be absent, or when present are much less severe.

There still remain to be mentioned the two organs of the body in which the process of embolism offers the most distinctive picture, to wit, the retina and the skin.

Embolism of the retina is marked by the occurrence of sudden blindness. The retina shows the typical picture of infarction in the extravasation of blood. Sometimes the whole process can be distinctly studied with the ophthalmoscope, and the diagnosis of a doubtful case has been established in this way. A septic embolus may develop panophthalmitis with destruction of the globe. Affection of one eye is usually followed by affection of the other. Litten especially described the changes in the retina which occur in acute malignant endocarditis. Hemorrhages and emboli may produce tinnitus aurium or may affect or destroy the hearing.

Occlusion of the vessels of the skin produces eruption of every character. Sometimes there is a typical roseola or an urticaria; sometimes the eruption resembles that of measles or scarlet fever; sometimes it presents the pustular form of variola or the larger bullæ of pemphigus. Most frequently the eruption takes the form of petechiæ or more extensive ecchymoses. In any case it may cover the whole body, though it is usually confined to, or most marked in, the lower extremities. In rare cases there may be sweating more or less profuse, and in still rarer cases furfuraceous or lamellar desquamation.

The characteristic bacteria which have produced the affection have sometimes been demonstrated in the blood or fluid taken from the eruption.

v. Ziemssen called attention to the fact that thrombus of the left auricle was followed in some cases by circumscribed gangrene of the lower extremities, which was announced by anæsthesia and extreme coldness of the extremities. The condition was not always dependent upon embolism but was sometimes due to arterial thrombosis with arrest of the circulation. Curious are those cases in this connection in which a thrombus is carried from the right to the left auricle through an open foramen ovale to lead to embolism in the course of

the greater circulation. Cohnheim and Litten reported cases of this kind, and Zahn distinguished this process as the paradoxic embolus. The occurrence of embolism of this kind is further illustrated in the following reports:

Henrot reported a case of vegetative endocarditis with purpura, phlyctenæ, and gangrene. The same author reported another case marked by purpura and anasarca, and a third case attended by purpura and attacks of intermittent fever. This case recovered.

Esquerdo reported a fatal case of septic endocarditis attended with embolism of the middle cerebral artery, of the axillary and of the popliteal arteries; Fenwick a case of septic endocarditis followed by axillary embolism and eventually by apoplexy; Barié and Du Castel a case of septic endocarditis of puerperal origin attended by obliteration of the aorta and the iliac arteries by an embolic clot, followed by paraplegia; Bengelsdorff a case of primary endocarditis terminating in embolic paralysis; and Ellis a case of septic endocarditis with embolism of the arteries of the left leg.

Emboli occurred in one-fourth of all the cases tabulated by Sperling, and of these 84 cases deposits were found in the kidneys 57 times, the spleen 39 times, the brain 15 times, the liver and alimentary canal 5 times each, and the skin 14 times.

Forms.—Septic endocarditis may show itself under a variety of forms, according to the localization of the septic products in the body. Such forms have been distinguished as the typhoid, pyæmic, pulmonary, rheumatic, cutaneous, cardiac, and meningeal.

The typhoid form shows the well-known picture of typhoid fever. The brain is distinctly clouded; there is hebetude, headache, adynamia, insomnia, states of coma or vigil, sometimes the characteristic combination of these conditions known as coma-vigil, and muttering delirium. The tongue is dry and coated, sordes is seen about the lips, teeth, and alæ of the nose, the abdomen is distended, there is diarrhœa, sometimes with a discharge of blood, the spleen is swollen, and the picture of typhoid fever is still more closely simulated in the appearance of sudamina and roseola upon the surface (Lancereaux).

The pyæmic form is distinguished by the frequent occurrence of chills, with profuse sweats, and by the abrupt elevations and depressions of temperature characteristic of the streptococcus curve. The pulse is quick and bounding, 140 to 160 per minute, is often irregular and intermittent. There is usually in these cases pronounced gastro-intestinal catarrh, and icterus is a common complication. Infarction of the liver is not uncommon among the localizations of emboli, and acute yellow atrophy has been observed in certain cases.

The pulmonary form is distinguished especially by the presence of cough and the predominance of dyspnœa. Bronchial catarrh and broncho-pneumonia are common complications, and the expectoration of frothy mucus tinged with blood indicates the development of infarction of the lungs. Pleurisy occurs also occasionally, sometimes independently and sometimes in connection with pneumonia as a product of invasion by the pneumococcus.

Wandering pains in the muscles, especially in the loins, are common manifestations of a septico-pyæmia, and most cases show more or less implication of the joints. Except in those cases which occur in the puerperium septic joint affections are comparatively rare in the course of endocarditis.

The cutaneous form is distinguished by the abundance and variety of eruptions upon the surface as the result of metastatic processes.

The cardiac form is distinguished by the predominance of palpitation, pain, and dyspnœa, symptoms which are to be referred for the most part to involvement of the myocardium.

Where the toxic or metastatic process more especially attacks the brain, the form is distinguished as cerebral, cerebro-spinal, or meningeal. In this case the symptoms may closely resemble those of cerebro-spinal meningitis. There is intense pain in the head, retraction of the neck, vomiting, strabismus, convulsions, contractions. These are really cases of cerebro-spinal meningitis which complicate the lesion in the heart. Sometimes the disease process begins with a pneumonia, intercalates an endocarditis, and ends with a meningitis. Netter and Weichselbaum repeatedly demonstrated the pneumococcus in the exudation of the meninges as well as in the lesion in the heart in these cases.

Diagnosis.

The diagnosis of endocarditis is determined by the subjective signs: chills, with fever, anxiety, pain in the region of the heart, palpitation, sometimes with headache, insomnia, and dyspnœa. In certain cases a change of posture from the recumbent to the semi-recumbent, to secure relief of pain and distress, excites the suspicion of the practitioner. The diagnosis is further determined by the physical signs: increased or decreased action of the heart, dislocation of the apex, increased dulness, and murmurs characteristic of a valve lesion; further by the effects of disturbance in the blood supply to distant organs, especially by the processes of embolism in the brain, lungs, spleen, and kidneys, characteristically in the retina and skin.

In all cases it is to be remembered that endocarditis is never a primary disease but occurs in the course of the infections, notoriously

in acute rheumatism, septicæmia, pneumonia, tuberculosis. Endocarditis is much less frequent in typhoid fever, erysipelas, gonorrhœa, diphtheria, and scarlet fever.

When the symptoms are present in characteristic array the diagnosis is easy. But these symptoms are by no means always present, so that the diagnosis is often difficult, and in case of infection of the wall of the heart away from the valves the diagnosis is often impossible.

As a rule, however, the valves are affected and in at least one-half of the cases the disease is concentrated about the mitral valve. Of the 300 cases tabulated by Sperling, the disease was found 255 times on the mitral, 129 on the aortic, 29 on the tricuspid, and 3 times on the pulmonary valves. The mitral valve was affected alone 157, the aortic valves alone 40, the tricuspid alone 3 times, the valves of the pulmonary artery alone in not a single case. In the majority of these cases the deposits interfered with the closure of the valve so that the signs were those of mitral insufficiency.

The heart sounds are sometimes weakened during the acute stage of the disease, occasionally to such degree as to become inaudible. Potain was so much impressed by this fact as to consider this weakening a sure sign of endocarditis. But the same weakening is often noticed in typhoid fever, and further the first sound especially is often rather lengthened and roughened before the characteristic bruit is developed (Sturges).

The great difficulty is offered by the so-called accidental murmurs. These occur usually in connection with anæmia. They are unattended by any signs of enlargement of the heart or by accentuation of other valve sounds. They are not found, as a rule, in connection with diseases which most frequently produce endocarditis; at least they are not found in the early history of these diseases. They are nearly always systolic. The pericardial murmur is more superficial, is more distinctly affected by change of posture and by pressure, and does not correspond so distinctly to the phases of the action of the heart. See also Sclerotic Endocarditis.

It is necessary to know, if possible, not only the existence but the character of the endocarditis, that is, whether the affection be simple or septic. Acute rheumatism usually produces the simple form, while the diseases of septico-pyæmia produce the septic form. To this rule there are, however, many exceptions in both directions. The septic form is distinguished by the frequency of chills or shivering fits, followed often by sweats and with the general signs of pyæmia. The patient often falls into a typhoid state. There is headache with hebetude, sometimes with muttering delirium. These signs, in con-

nection with the dry tongue, sordes, enlargement of the spleen, roseola, and diarrhœa, present the distinct aspect of typhoid fever. But the temperature curve is not typical as in a case of typhoid fever. The fever is not sustained uniformly at a high level for so long a period of time as in typhoid fever. Moreover, typhoid fever shows none of the physical signs of endocarditis, as it is one of the diseases which rarely affect the endocardium. Sometimes the scale is turned by lighter points. For instance, icterus speaks for endocarditis and against typhoid fever.

In pyæmia it is only a question whether the condition affects the heart. This question may be answered only by the presence or absence of physical signs, that is, by the presence or absence of a bruit, of enlargement of the heart, accentuation of valve sounds, etc., etc.

Tuberculosis is often differentiated with difficulty. Miliary tuberculosis is usually found in connection with localized deposits, with evidence sometimes of enlarged bronchial glands, sometimes with a previous history of bad health, sometimes with cough and expectoration of matter in which the tubercle bacillus may be detected. Usually, however, cough and expectoration are absent in miliary tuberculosis. But in this disease there is a continuous high temperature with rapid consumption of the tissues. Septic endocarditis shows the see-saw rise and fall, which constitutes the so-called "streptococcus curve." This is a point, however, upon which no stress can be laid, as acute tuberculosis is characterized in the same way. Phthisis florida is caused by septic invasion of the blood and shows the same characteristic streptococcus curve.

In rare cases the discovery of tubercles in the choroid coat determines the diagnosis, or the occurrence of hemorrhage from embolism establishes the existence of endocarditis. In cases not too acute the diagnosis may be established by means of tuberculin.

A point of distinction between the simple and septic forms may be learned from the character of any embolic process which may develop. Emboli from the simple form are bland and produce the symptoms only of mechanical occlusion, whereas emboli from the septic form are infected and produce metastatic abscesses. The separation of simple and septic forms may further be determined by examination of the blood.

To make this examination the finger of the patient is disinfected with soap, alcohol, sublimate, and ether. The lancet is purified by fire by being drawn through the flame of a Bunsen burner. The end of the finger is then punctured and the blood received upon a platinum needle previously heated to a white heat and cooled. Cultures may

now be obtained in test tubes, or preferably upon plates. Where it is necessary to make more extensive observations, larger quantities are drawn by aspiration with a sterilized syringe from one of the veins in the arm, which is made to swell by compression above, as in the operation of venesection. The small instrument with the exceedingly fine tube, devised for intravenous injection, answers admirably for this purpose.

Hitherto these examinations of the blood have not been very satisfactory, partly because they were not made at the proper time and partly because the quantity withdrawn was insufficient. Pyogenic micro-organisms most abound in the blood during the period of chill or in the course of the fever which immediately supervenes. At other times they are literally few and far between, and this is found to be the fact especially in cases which are of malignant character, so that clinical diagnosis has hitherto received but little aid from bacteriological studies of endocarditis. But the examinations of the blood in pyæmia have recently been rewarded with such uniform results as to encourage further investigations in every field of these affections. Thus Sittmann, who made his examination by mixing blood with fluid cultures and spreading the mixture upon plates, obtained positive results in every one of 23 cases. This examination disclosed the staphylococcus 11 times, the streptococcus 4 times, the pneumococcus 6 times, and the staphylococcus and bacterium coli 2 times. The number of bacteria varied greatly; the staphylococci ranged from 1 to 14 per cubic centimetre of blood, the streptococci from 17 to 2,200. Septico-pyæmia always showed the presence of pyogenic micro-organisms, even though in small quantity, circulating in the blood. The bacterioscopic examination of the blood is therefore the surest means of reaching the diagnosis of septico-pyæmia.

Sometimes characteristic bacteria may be found in the excretions, so that the saliva, the sweat, and the urine should be examined bacteriologically. The various cutaneous lesions or metastatic abscesses may be incised under the precautions of asepsis and cultures may be made from the fluid withdrawn in this way. Claisse once found pneumococci in certain spots of purpura in endocarditis, and Leube once demonstrated characteristic bacteria in metastatic pustules of the skin. These examinations have not only disclosed the micro-organism but have revealed the site of primary deposit. Thus the pneumococcus would refer to a process in the lungs, the bacterium coli to an affection of the intestine or of the bile ducts, or to cystitis, the staphylococcus to an osteomyelitis, the streptococcus to some manifest process of suppuration or to some cryptogenetic source, which may be discovered on careful search. Finally, the nature of

the micro-organisms may be established by inoculation experiments upon the lower animals.

It is interesting to know whether the attack is recent or whether the present attack is an acute exacerbation of a chronic condition. Acute is separated from chronic endocarditis, that is, the simple and septic from the sclerotic forms, by the presence of fever in the acute cases, and by the gradual extension of the inflammatory process, as from valve to valve. The existence of a chronic endocarditis which may have been hitherto unsuspected may be sometimes disclosed in the history of the case; that is, a careful inquiry may elicit the fact that the patient has had for some time occasional attacks of palpitation, dyspnœa, or other sign of insufficiency when unusual demands have been made upon the heart. The question whether the present attack is to be interpreted as an exacerbation or a relapse may be determined only by knowledge of the previous history of the case. Sometimes the records of life insurance examinations, when made by competent hands, furnish information of value in this regard. Examinations for military service are of more value where this service is general, as in Europe.

Sometimes the nature of the lesion sheds light upon the case. Thus a soft blowing murmur at the mitral valve, heard in greatest intensity at the apex and synchronous with the first sound of the heart, may be referred more particularly to an acute endocarditis with muscular paresis; while a presystolic murmur indicating stenosis would refer more especially to a chronic case. Acute endocarditis is more apt to affect the papillary muscles in such a way as to weaken them and lead to relative insufficiency. Affections of the valves at the base belong more frequently to chronic endocarditis and to atheroma. Affection of the right side of the heart, when not congenital, is also more characteristic of extension of endocarditis under a longer duration of the disease. The murmur of relative insufficiency at the tricuspid valve, which belongs to the stage of broken compensation, is also more indicative of a chronic process.

Thus it is seen that a mere diagnosis of endocarditis will not suffice for the requirements of the present day. It is necessary also to determine the character of the affection, whether simple, septic, or sclerotic, and, as far as may be, the nature of the originating cause.

Prognosis.

The prognosis varies greatly, but in no case may it be said that endocarditis is a trivial disease. It is true that in many cases the disease is latent, but even latent cases are liable to complications

which give them gravity. In the most quiescent case a thrombus secreted between the trabeculæ may be washed into the circulation and even cause death by occlusion of an important artery in the brain. Inflamed chordæ tendineæ may suddenly give way to produce at once the signs and lesion of a complete insufficiency. Fortunately such accidents in these cases are extremely rare and are reported as curiosities in medical literature. In most cases of simple endocarditis the prognosis is good. Under favorable circumstances hypertrophy sets in and compensates, at least for a long time, any lesion of the valves. The prognosis will be determined in these cases largely by the character, extent, and situation of the lesion in the heart, more especially by the degree of the compensating hypertrophy. A case of simple endocarditis is grave from the fact that one attack renders the individual more liable to another attack. The micro-organisms which cause the disease are somewhere secreted in the body and under some exciting cause they make renewed attack upon the heart.

The prognosis in a simple case is further affected by the number and situation of the embolic processes. Embolism of the brain is more serious than embolism of the spleen, but even in the brain the effects will vary according to the localization. Thus Goldscheider reported the case of a woman, aged thirty-one, in whom death was caused by an embolus in the basilar artery. Leyden reported a similar case. The most common lesion, embolism in the arteries of the Sylvian fissure, leaves a hemiplegia with aphasia. On the other hand, Raynaud reported two cases of infarction of silent centres in the brain, discovered of course only upon autopsy. O'Carrol reported a case of sudden death from embolism of the coronary artery.

More grave is the prognosis in the case of septico-pyæmia. The nature of the originating malady plays an important rôle here, and the form of the disease or the other localizations of septico-pyæmia make a difference. Thus the meningeal and pyæmic forms are most dangerous; the typhoid form is very grave; the pulmonary and cutaneous forms may be much less serious.

A few bland emboli may not make much difference, but the multiplication of infarctions indicates a grave case. For the most part the sensorium is clear, but the presence of cerebral signs, especially of comatose states which might indicate meningitis or encephalitis, make the outlook ominous. Icterus gravis and hemorrhagic nephritis give gravity to the prognosis. The prognosis is more favorable in infancy than in youth. All the signs of endocarditis, including especially the murmur, may disappear in early life and there may be an entire *restitutio ad integrum*.

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In any case the intensity of the character of the murmur does not necessarily indicate the extent or intensity of the inflammatory process. The character of the murmur depends rather upon the situation of the lesion and the interference with the action of the valves. Murmurs which come and go, which vary in intensity, belong rather to loose vegetations which change position, and floating vegetations are always more or less dangerous. The intensification of murmurs previously present and the appearance of new murmurs indicate extension of the disease and are therefore unfavorable signs. Musical murmurs attended with fever have a very unfavorable prognosis (Rosenbach). Murmurs which disappear never to return have a more favorable prognosis, provided always that the thrombus which may have caused the murmur has not blocked an important vessel in some distant organ. In the absence of embolism the disappearance of murmurs is attributed to resolution of the process of inflammation or to repair of tissue, for instance, to closure of a fissure or rupture by a coagulum which subsequently becomes organized. These signs and results have been observed in experiments on the lower animals where the valves have been artificially injured. In these experiments it is seen that the action of the heart becomes tumultuous for a time under the reflex excitation of the endocardium, and that the characteristic bruit may be observed only after the subsidence of the period of excitement. These conditions may receive therefore their proper interpretation in prognosis.

Pregnancy and the puerperium not only invite the attack in the first place, but aggravate it when it has already occurred. Women who have once suffered are liable to renewed attack at these times.

Finally something of the prognosis may be determined by a knowledge of the nature of the cause as revealed by examination of the blood. Simple endocarditis has, as stated, barring accidents and complications, for the most part a favorable prognosis. In septic endocarditis the prognosis of an individual case is influenced by the various factors just described. Septic endocarditis has in a general way the prognosis of septicæmia, of which process the endocarditis is to be regarded as a localization in the heart. The prognosis of septicæmia, as determined by the cause thus revealed, becomes evident from the results of the studies of Sittmann, mentioned above. Of the 23 cases, 16 ended fatally. The fatal cases included all the streptococcus infections, 6 of the 11 staphylococcus, 4 of the 6 pneumococcus and the mixed infections.

So long as endocarditis remains free of complications, especially of embolic processes in the lungs, gangrene of the lungs, suppurative pleuritis, and sinus thrombosis, the prognosis is not very grave.

Even the supervention of these grave complications does not make the prognosis necessarily fatal. Here, too, much depends upon the localization of the lesion. Thus Fraentzel declares that he never knew a case of septic endocarditis complicated by thrombus of the sinus petrosus or a pylephlebitis which did not terminate fatally. There is no doubt, however, that cases do recover from uncomplicated septic endocarditis marked by such distinct and violent chills as to simulate intermittent fever. Cautley reported a recovery in such a case.

Treatment.

The treatment of acute endocarditis resolves itself into address to the underlying and originating malady and relief of the symptoms on the part of the heart. The most essential thing is rest. For the most part the rest is secured by the fact that the patient is already confined to bed by the original disease. Where the process develops more insidiously rest is equally important in the acute period of the disease. Endocarditis affects the valves because of their activity. Rest of the body and rest of the heart gives comparative rest to the valves. The first infection of the endocardium is usually attended by excitement of the heart. This excitement should not be increased by exercise of the body. The nervous mechanism of the heart is an exceedingly fine and complex regulatory apparatus, which enables the heart to adjust its action with the greatest nicety to the wants of the body. It enables the heart also to adjust itself to its own needs in every particular. Any outside interference with this process is likely to be meddlesome and mischievous. During this period the heart should be let alone. Irreparable damage may be done by overstimulation at this time.

The heart is spared extra effort also by care as to the diet and by attention to the secretions. Milk is the best food. Where milk is objectionable it may be substituted by malted milk. In the presence of fever, especially in connection with disturbance of the stomach, the lightest diet will suffice. Milk may be diluted one-half or one-third with Seltzer water. The diet may be restricted for a few days to the gruels of farina or oatmeal. The room should be well ventilated, the body should be lightly but warmly clothed that the lungs and skin may throw no extra work upon the heart. The patient should be secluded as far as may be in a quiet room, that the emotions may not be excited or fatigue experienced from the intrusion of perhaps well-meaning but ignorant and officious visitors.

Address to the originating malady applies especially to rheumatism. The salicylates have almost a specific control over this disease. Of this fact there can be now no doubt, though the mode of action of

the remedy is not at all clear. So long as the true nature of rheumatism is unknown, it is idle to speculate concerning the action of remedies used in its relief. Meanwhile we may be satisfied with the clinical results. For many years there was much idle speculation concerning the action of quinine in the cure of malaria. So soon as the nature of malaria was determined, experimentation became direct, and it was soon ascertained that quinine kills the cause of malaria in the blood. Whether the salicylates control rheumatism by reason of antimycotic properties or whether they act as antitoxins cannot be declared at the present day. The fact that the administration of the remedy must be continued for some time to prevent recurrence after the first effects of the disease shall have been controlled speaks for a direct parasiticidal action. As in the case of syphilis and malaria, it would appear as if the cause of the disease were secreted to make irruption from time to time into the blood, and as if, after immediate effects have been obtained, immature stages of development refractory to treatment still remain in the body. As these stages reach maturity the remedy must be used anew.

Rheumatic endocarditis calls, therefore, for the use of the salicylates to jugulate or curtail the rheumatism, or at least to mitigate the severity of the individual attack. Unfortunately, the salicylates depress the action of the heart. They must, therefore, be used with caution, and the extreme doses which may be demanded in the treatment of acute rheumatism, both to alleviate the suffering and prevent the extension of the disease, are not justifiable when the complication on the part of the heart has already set in. The salicylates are, therefore, of more value in the prophylaxis than in the treatment of endocarditis. Nevertheless some of the salicin compounds may be used in the acute stage of endocarditis, especially in the presence of fever, and they are actually indicated in cases of renewed attacks of rheumatism to prevent the extension of disease in the heart. So, too, the salicylates are distinctly indicated when it is known that the disease is produced by the pneumococcus. The dose of the sodium salicylate, for instance, may be graduated from five to ten grains every two to four hours during the acute stage of the disease in the presence of fever. Anything like an excessive dose punishes the patient by weakening the action of the heart, and by producing profuse sweats which may lead to a dangerous collapse. To prevent these accidents it is advisable, therefore, to administer the salicylates with some aromatic tincture or in connection with a teaspoonful of good whiskey or brandy and to stop short always of toxic signs, ringing in the ears, nausea, vertigo, etc.

A moderate amount of palpitation may be let alone as part of the

self-adjusting action of the heart. Should the palpitation become distressing or the action of the heart grow too tumultuous, it may be controlled safely and not unpleasantly by the application of cold, especially in the form of compresses wrung out of cold water. Sometimes a mild dose of phenacetin, grs. iii.–v. at bedtime, quiets excessive action and secures restorative sleep.

Any ordinary amount of pain may be relieved in the same way by a small dose of phenacetin, or, if more severe, by broken doses of Dover's powder, two or three grains, repeated, if necessary, every two or three hours. The latter may be conveniently given to a child in the form of a syrup. It would be a very exceptional case of endocarditis in which the pain would be so severe as to call for the use of morphine.

Stimulants are to be used with caution. In the presence of dyspnœa, cyanosis, coldness of the extremities, resort may be had to the use of ether in the dose of gtt. v.–xv. or of that preparation of ether known as Hoffmann's anodyne, in double this quantity. Sometime a teaspoonful of brandy quickly restores a failing heart. A cup of black coffee may have the same effect; or the coffee may be fortified with brandy. A cup of strong tea with a teaspoonful of rum may be a substitute for those who prefer tea. In the presence of collapse or of heart failure, restoration may be more quickly reached by the subcutaneous injection of caffeine grs. ii.–iii., especially of the sodium benzoate or of the sodium salicylate of caffeine. A broad, thick sinapism made of fresh mustard and warm water and applied over the whole precordial region is not to be despised in these cases. A flagging heart is better held up in the long run by digitalis in the form of the tincture gtt. v.–x. every two to four hours. This remedy is, however, to be used with great caution and good judgment in acute endocarditis. Digitalis is a sharp sword which cuts both ways. It may easily overstimulate a heart struggling to regulate itself.

In all cases of septic endocarditis search must be made for the cause of the infection, that the endocardium be not continually fed from some septic centre. Thus the scientific treatment of endocarditis may resolve itself into the operation of curetting a case of endometritis, of treatment by deep injection of an internal urethritis, prostatitis, or cystitis; of treatment of tuberculosis of the lungs by climate, creosote and tuberculin; of treatment of osteomyelitis by the exsection of suppurating bone, etc., etc., etc. In the absence of any specific address to the cause of the disease, it is wise to confine the actual treatment of acute endocarditis largely to the recumbent posture, the application of cold, and the relief of pain.

For the rest the case may be resigned to time, awaiting the discovery of specific control of the products of sepsis, perhaps through serum therapy.

Sclerotic Endocarditis.

(Atheroma; Chronic Endocarditis; Chronic Valvular Disease.)

History.

Our knowledge of chronic valvular disease antedates that of acute endocarditis. The earliest anatomical observers could not fail to recognize the gross changes produced in the valves by deformities in their structure or by the more or less extensive deposits of fibrin upon their surface. As already stated in the notes on the general history of heart disease, valvular lesions were recognized by the old French and Italian anatomists. Vieussens described cases of sclerosis with insufficiency of the mitral valve, and Lancisi and Albertini saw dilatation of the cavities of the heart. Sénac (1749) wrote an elaborate work on the diseases of the heart, and while his work was accepted by the great masters Morgagni, Haller, van Swieten, and Pringle as the highest authority in its day, it failed, because based upon pathological data, to find general appreciation. There was still too much of a tendency to construe prominent symptoms, dyspnœa, dropsy, etc., into independent diseases.

With the discovery of percussion by Auenbrugger (1761) and the general dissemination of this discovery by Corvisart (1811), it became possible to recognize the hypertrophy which sets in in the course of valvular disease, and in 1814 Kreysig, of Dresden, devoted especial attention to inflammation of the lining membrane of the heart. The knowledge of the relation of the symptoms to the lesions in the earlier decades in our century is indicated by a report by v. Rhyn, which reads: "Hydrops universalis; palpitationes cordis; summa anxietas; mors; cor dextrum hypertrophycum; in ventriculo sinistro ossificaciones valvularum."

Laennec (1828) illuminated the whole field with the stethoscope, and made it possible to localize by demonstrations of almost mathematical accuracy the particular valve of the heart affected—not only that, but the particular character of the affection, so that scarcely any disease in the body may be so precisely diagnosticated as lesions of the valves hidden deep in the interior of the body of the heart.

Bouillaud (1840) now established more accurately the causative relation of rheumatism, and Hope, Williams, and Andral made important contributions to prognosis and treatment. Accurate clinical

observations were furnished also later by Andral, Piorry, Corrigan, Chomel, Latham, Walshe, and especially by Stokes, whose celebrated work on "Diseases of the Heart and Aorta" was published in 1854. Andral describes the process of thickening, induration, shortening of tendons, vegetation, destruction of the free border of the valves, perforation, rupture, etc., with astonishing accuracy. Stokes, who dwells upon the symptomatology, still does not separate the stenoses and insufficiencies; but Friedreich (1865) sharply defines the two conditions. Rokitansky (1845) made numerous contributions from his immense field in pathological anatomy, and Skoda, a few years later, refined the subject of diagnosis by auscultation to the utmost possible limits. Traube (1856) made especially plain the connection between the diseases of the heart and kidneys to which Bright, as long ago as 1827, had called attention in his epoch-making work on the subject of kidney diseases. Subsequent contributions, concerning especially the field of etiology, have reference to the relation of the infections which have already been studied in connection with acute endocarditis.

Etiology.

Acute endocarditis may subside and the lesions may disappear to leave no trace. But this fortunate termination is not the rule; on the contrary it is rather the exception. As a rule the inflammation becomes chronic, or at least the lesions which have been left remain to produce permanent change in the endocardium, and thus permanently interfere with the action of the valves.

The most common cause of this change is the inflammation which ressult from infection and which has been studied under the head of acute endocarditis. Chronic endocarditis by no means, however, always results from the acute form of the disease, or, at least, it may be said that endocarditis develops at times so insidiously as to have escaped observation in the acute stage.

Proof of the septic nature of this endocarditis is furnished in the general infection which sometimes occurs in the course of the sclerotic form. Thus Paulus reported the case of a woman, aged twenty-seven, who had suffered from a mitral lesion for eleven years and was then seized without any known cause by a relapse of acute endocarditis with septico-pyæmia which destroyed life in the course of five months. In this case the Staphylococcus albus was cultivated from the vegetations on the valves. In a second case a man, aged fifty, died of sepsis which had developed from a valve lesion, the existence of which had not been appreciated by the patient. The

streptococcus was cultivated from old vegetations on the valves in this case.

Further, not all cases of diseases of the valves by any means proceed from endocarditis. Alteration of structure and interference of action are caused with great frequency also by atheroma, that is, by those processes which in the arteries are distinguished as arteriosclerosis. These processes are not considered as inflammatory; they are, more strictly speaking, degenerative processes. They are even more insidious in their onset and development. Although they are considered as degenerations they may not be separated from the infections, but they belong rather to the chronic than the acute infections. So endocarditis is known to be an expression of an acute infectious process, while atheroma may be said to be a result of a chronic infectious process.

The influence of particular diseases and the relation of the various diseases in the development of this complication have been sufficiently studied under acute endocarditis. Thus it has been shown that acute endocarditis in its simple form occurs most frequently in connection with acute rheumatism, pneumonia, tuberculosis, while endocarditis of septic form may result from any of these processes, but does occur more frequently in connection with the various infections of septico-pyæmia. In most cases the endocarditis, whether of simple or septic form, may be distinctly recognized in the course of, or as a sequel to, some of these infections, so that when the condition becomes chronic in the heart the nature of the affection will have been already established. It is known then that chronic endocarditis develops in certain cases directly from the acute form of the disease, while in other cases it is introduced by the same causes more insidiously and is recognized only when the disease has already become chronic. The majority of cases of chronic endocarditis develop from the lighter form, for the simple reason that most cases of the lighter form survive, while most cases of the septic form succumb. Chronic valvular disease, in so far as it results from endocarditis, shows the lesions which belong to the simple form of the disease or the conditions which result from subsequent change in these lesions.

But chronic valvular disease by no means depends exclusively upon endocarditis of any form. Characteristic lesions are produced, also, as stated, by atheromatous changes. Montault, as long ago as 1831, reported a case of inflammation of the internal membrane of the heart in a patient aged seventy-three, which reads much like the appearance of an atheromatous ulcer. The symptoms were those of asthma and chronic bronchitis. On autopsy a remarkable alteration

was found on the internal surface of the cavities of the heart, which were "covered with a deposit of a matter like pus."

The factor of highest importance in the development of atheroma is age, so that the lesions of the valves which result from endocarditis are discovered more frequently in youth, the period of the prevalence of the acute infections, while the diseases of the valves, which result from atheroma, occur most commonly in advanced life. Atheroma is a process which shows itself most frequently in the aorta and extends thence by mere continuity of structure to involve the aortic valves. Endocarditis, as has been shown, expends itself mainly upon the mitral valves, so that affection of the mitral valves is more common in youth and of the aortic valves in age. It is needless to state that either process may extend from one set of valves to the other and that in the more exceptional case the changes of endocarditis may be found in the aortic valves, while the changes of atheroma may be found in the mitral valves. So lesions of the mitral valves are found especially between the ages of ten and thirty, of the aortic valves between the ages of thirty and fifty years.

Endocarditis, as already stated, may be purely mural, but it is only in the exceptional case that the valves escape. On the other hand, atheroma is more frequently confined to the aorta or other arteries. The escape of the aortic valves in individual cases under the process of atheroma is counteracted to a considerable degree by involvement of the vessels of the heart itself in the atheromatous change.

Atheroma is a peculiar hyaline, fatty, and chalky degeneration which extends from the aorta to involve the valves at the base of the heart. The process is the same in essence as that of arteriosclerosis in the smaller vessels. In fact atheroma is said to be the arteriosclerosis of the aorta and heart. Atheromatous change is always found somewhere in the body in advanced life, and is not infrequently precipitated by the conditions which hasten the changes of old age, especially by alcoholism, syphilis, hard work, lead-poisoning, and gout. Thus it is sometimes present at a period as early as the age of thirty-five years.

In the study of the etiology of chronic valve disease, mention at least must be made of the effect of the strain which the valves suffer under increased pressure of the blood. The effect of strain is especially to be observed in cases of prolonged muscular effort and as the result of arteriosclerosis, Bright's disease, etc. Trauma may be the final cause of valve lesion. Thus a powerful muscular effort may lead, under the strain of distention of the heart, to rupture of a tendinous cord or severance of a valve from its connection at its base.

Such an accident is only likely to occur when the cord or valve structure has been weakened by previous disease, as by an attack of endocarditis. It is easy to understand how the rupture of an aneurysm might lead to the same incompetence. Aside from the influence of toxins, which may directly induce degenerative change or indirectly interfere with the nutrition of the endocardium, the valve may be stretched in places or suffer solution of continuity, and the lesion may be repaired by cicatricial tissue. Such cicatrization, scleroses, or fibroid degenerations invite the localization of endocarditis, become the seats of colonies of micro-organisms or deposits of fibrin from the blood, or more readily undergo that subsequent hyaline, fatty, or chalky degeneration which constitutes the atheromatous process.

Most cases of endocarditis leave permanent lesions; all the cases of atheroma leave lesions which are permanent, so that through one process or the other valve diseases are not infrequent. But statistics vary greatly according as they are derived from revelations in the dead room or from diagnoses at the bedside. Valve disease is often latent in life and in many cases is recognized only upon the post-mortem table. Thus Dittrich finds valve disease in 5 per cent., Förster in 11.3 per cent., Chambers in 17 per cent. of cases. Raynaud made an average of 11 per cent. from different statistics including 7,347 autopies. Duchek, who made his estimate from bedside examinations, put the proportion at 2.4 per cent. Sex makes but little difference. Atheromatous changes in the aorta are more frequent in the male sex. Affection of the mitral valve is said to be more frequent in the female sex.

Heredity is an essential point in the etiology of all forms of heart disease. It is noticed that heart disease occurs with especial frequency in certain families. Not that any particular form is transmitted or that the disease is especially congenital, but that there is a disposition in the greater excitability of the nervous system, the heart being influenced by lighter irritants. The tendency to arteriosclerosis is certainly distinctly transmitted. Moreover, certain families are especially predisposed to rheumatism and some of them to grave forms of the disease. Then certain individuals affected with rheumatism show no disposition to endocarditis. On the other hand, in other families the disposition is distinct. In some families attacks of scarlet fever and diphtheria are followed by heart disease more commonly than in others (Rosenbach). Venturi details the symptoms of heart disease in two cases, sisters whose mother and aunt (mother's sister) had died of the same affection. In both the ancestors there was combined affection of the mitral and aortic valves, and also stenosis

at the tricuspid orifice which was evidently a congenital lesion. Schott elicited in 245 patients affected with valve lesions affection of one or several members of the same family, in the ascending or descending line, 58 times.

As to the frequency of affection of the different valves, later observers only substantiate the statistics furnished by Ormerod (1851) who found on autopsy in 801 cases of chronic valve disease:

Age.	No. of Cases.	Mitral.	Aortic.	Tricuspid.	Pulmonary.
Under 20 years	30	27	17	2	0
From 20 to 30 years	31	26	14	3	3
" 30 to 40 "	45	24	27	5	2
" 40 to 50 "	32	25	22	3	1
" 50 to 60 "	24	15	22	4	0
Above 60 years	19	16	14	0	0

Statistics of later date have been abundantly cited under acute endocarditis. Here may be mentioned for the sake of completeness the multiple collection of Parrot, who found valve lesions in the right heart in but 57 of 1,058 cases. The relative frequency of affection of the various valves was as follows: Mitral 621, aortic 380, tricuspid 46, pulmonary 11.

Morbid Anatomy.

The changes which the valves undergo in endocarditis have been sufficiently described in the chapter on acute endocarditis. The processes become more extensive, and in many cases additional deposits and retrograde changes, marked especially by shrinkage and induration, distinguish the chronic form. The cell tissue becomes thicker and more opaque. The thrombi which constitute the mass of the vegetation become condensed, lose some of the reddish color, grow more gray or white, and lose something of the exuberative verrucose or cauliflower appearance of the fresh deposit to become more dense and fibroid in character.

It is the fibroid degeneration which especially characterizes the chronic indurative change. The connective tissue, which has increased by proliferation, now undergoes shrinkage, and the process is facilitated by the fact that the vessels become obliterated and thus hasten the retrogressive change. The lines of junction at the closure of the valves, being subjected to the greatest strain, especially undergo this fibroid change. Thickening begins in this region sometimes to traverse the valve as a tendinous cord, but soon to extend to involve a greater or less amount of all the tissue of the valve. Under the process of cicatrization, contraction takes place, the valve shrinks

and is no longer able to close the orifice. This incompetency is more especially hastened by the fact that the fibroid change involves also the muscular tissue as at the auriculo-ventricular orifice. The muscular tissue in this region may, therefore, no longer contract the orifice from the circular or ovoid to the more slit-like opening necessary to effect perfect closure. Moreover, the same process involves the chordæ tendineæ, which also become thickened and shrunken, and may thus forcibly restrain the valves from perfect closure. Not infrequently apposed valves become agglutinated so as to prevent their perfect adaptation to each other or to the sides of the heart. The mitral valves may in this way be converted into a funnel whose sides are more or less adherent to the wall of the heart or bound down by tough, inelastic tendinous cords.

The atheromatous process subsequently undergoes necrotic change, and the surface, which was at first covered with a tough yellow plate of hyaline or even chalky matter, is now changed into an ulcer filled with granulo-fatty diffluent matter, which has given the atheromatous process its name. Where this necrotic process is extensive, it may cut through the whole substance of a valve to lead to rupture or perforation, or, as the attenuated tissue yields under the strain of the blood pressure, to constitute aneurysm of the valves. The same atheromatous process may affect the substance of the heart or the aorta, or it may affect the columnæ carneæ or tendinous cords and lead to their rupture or destruction.

But the process of necrosis need not necessarily ensue. It is at all times more frequent in cases of septic attack under the influence of micro-organisms. Sometimes the tissue is substituted particle by particle with deposits of the lime salts, and thus extensive plaques or plates may be developed in the valves, especially in the aortic valves, whereby the valves undergo a process of calcification. Such deposits may be seen upon either surface of the valve, more especially, however, upon the aortic surface where the valve bears the strain of blood pressure, sometimes, however, extending through the whole substance of the valve to convert the entire segment into a rigid mass which can no longer be pressed against the sides of the aorta. The tip of the little finger may then be introduced behind the valve, which is felt to be an unyielding, rigid cup of bone or stone.

All these processes represent various stages of retrograde degenerative change, which takes place in tissue under interference with the processes of nutrition. The initial changes, therefore, are to be observed in the blood-vessels. The intima of the blood-vessels especially becomes thickened, the nuclei multiply, sometimes with development of round cells in the wall of the vessel, and thereupon

gradually supervenes the hyaline degeneration under which the wall of the vessel becomes thickened and the lumen contracted. These changes are to be found in all the vessels in every region of the heart, most markedly in the inner layers of the left heart where the processes of endocarditis or atheroma are most pronounced.

But these changes in chronic valve disease are not confined to the endocardium. In all cases disease processes are found in all parts of the heart. There may be no macroscopic but there is always microscopic evidence, in accumulation of round cells and the implication of the smaller vessels, of more or less pericarditis, and in all cases there is alteration in the muscle substance of the heart itself. The protoplasm of the muscle fibre is found in every stage of inflammation and degeneration, from accumulations of granular matter with abundance of pigment to substitution by fat granules and hyaline degeneration.

The heart muscle fibres show an albuminoid opacity which gives to the heart muscle a peculiar grayish-yellow color and a faint lustre preceding the marked changes of fatty degeneration. The muscle cells appear as if dusted over; the striation becomes less distinct. It is especially the papillary muscles and the trabeculæ which show these lesions. The peculiar condition elsewhere mentioned as "fragmentation," first described by Tedeschi, if present anywhere is shown always in the papillary muscle of the left heart. Oestreich insists that the separation of fibres which becomes apparent on the surface of section does not take place at the line of the cement substance but traverses the cell body, leaving the nucleus on one side, and he holds with v. Recklinghausen that the condition is a preagonal phenomenon. But Aufrecht is not willing to so consider it, as he saw it in most exquisite form in a case of dilatation of the right ventricle without other discoverable cause. Browicz considers the condition to be the immediate cause of death. Israel was unable to produce it in his experiments of subjecting the left ventricle to high pressure with mercury, attributing it to a lesion in the musculature such as is seen in pigment atrophy. Israel repeatedly found fragmentation sharply limited to disseminated areas of fatty degeneration.

Changes in the ganglia, besides those already described, were found by Kusnezow in seven cases of acute endocarditis from various causes and in fourteen cases of chronic valve disease. These changes, which were quite uniform, were the occurrence of round cells between the nerve cells and within the capsule of the ganglion cells, swelling and cell proliferation of the endothelium of the capsule, and albuminoid and fatty degeneration of the protoplasm of the ganglion cells. The disease process extended directly out from the valves to the

annulus fibrosus. The superior ganglia were affected partly from a pericarditis or an acute inflammation of the walls of the pulmonary artery such as is said to occur in pneumonia, and partly, as in the cases of sepsis, by embolism (Roether).

These degenerative changes result from defective nutrition under the action of toxins from the infectious processes themselves, and under mechanical arrest of the circulation from sclerosis of the coronary vessels. The heart substance wastes under these processes, pigment matter accumulates, and the heart shows the changes of brown atrophy. Side by side with these processes may be observed fibroid degenerations in the form of interstitial sclerosis and changes of muscular tissue into amyloid matter. The processes of arteriosclerosis and atheroma may entirely obliterate branches of the coronary artery and lead to softening of the muscular structure of the heart, myomalacia, which may in turn eventuate in aneurysm and rupture.

The connective tissue undergoes a distinct hyperplasia and accumulates in masses, especially about the vessels. With the cicatrization of this tissue the heart shows the signs of a genuine cirrhosis originating in the peri- and endocardium. These changes in the muscle structure of the heart itself account for the differences in the symptomatology of valve lesions. The incompetency of the heart, as will be seen, does not depend so much upon the valve which is affected or even upon the extent of the affection of the valve, as upon the condition of the heart muscle.

Further changes are detailed under myocarditis, hypertrophy, and fatty degeneration.

The ordinary means of determining insufficiency or stenosis post mortem are defective and unreliable. The method of determining competency at the autopsy by pouring in water proves nothing definite, for valves that might close under such conditions may remain open under the greater pressure of the blood in life or in the presence of dilatation of the aorta. Insufficiency of the mitral valve, for instance, is usually established on autopsy by opening the left auricle and pouring into it a certain quantity of water. The water penetrates to the left ventricle under the influence of gravity; the ventricle is then seized in both hands and firmly squeezed. Insufficiency is considered to be established by the fact that the water passes back in the auricle instead of issuing entirely into the aorta. Another way is to introduce a quantity of water into the left ventricle by means of a syringe or elevated funnel with a rubber tube. Insufficiency is established in the same way by reflux of water into the auricle.

But the penetration of water in the dead heart does not prove incompetency in life, as the conditions in life are very different. The

muscular tissue of the left ventricle forms a kind of sphincter which is an extremely important factor in the closure of the valves, and there is no knowing after death the capacity of the muscular tissue, the power of the papillary muscles, the true size of the cavities, etc. Stenosis may be more easily decided because of the stiffness of the valves and apparent narrowing of the passage. But a valve may be thickened in high degree and may still have remained competent and shown no signs of insufficiency of any kind in life. The difficulties are even greater with the aortic valves, for entirely calcified valves may have shown no signs of insufficiency in life while relatively perfect valves may have shown distinct signs. Somewhat more satisfactory is the method by measurement which may reveal a dilatation of the orifice. The circumference of the mitral orifices is in man 122 mm., in woman 93 mm.

The capacity of the left ventricle was determined by Legallois, by the amount of mercury by weight which it would contain, at 10.68; that of the right ventricle at 11.72. Bizot determined the size of the left ventricle as 78 mm. in height by 122 mm. in breadth, while that of the right was 84 mm. in height and 85 mm. in breadth. The fact that the right ventricle had a greater capacity than the left was known to Hippocrates.

The ventricles in the healthy adult during quiet action discharge about 250 c.c. per second, or 15 litres per minute. This quantity is discharged by the left ventricle into the aorta and by the right ventricle into the pulmonary arteries. Compensation is said to be perfect when, notwithstanding the defect, about the same quantity of blood is discharged as before.

The general outlook of a case of valve lesion may be appreciated from the character of the affection of the heart muscle, as proliferations of connective tissue are usually of progressive character. The arhythmia, dyspnœa, and early exhaustion, as well as some of the toxic symptoms which show themselves in heart disease, may be accounted for by the nature of the changes in the heart muscle itself.

The heart muscle is found in valve lesions in a state of hypertrophy which is sometimes extreme. In fact, there is no disease in which the enlargement of the heart becomes so great. It is easy to understand the hypertrophy which develops in the face of an obstacle. It is the same as that which occurs in any hollow muscular viscus, as, for instance, in the stomach behind a constricted pylorus or in the bladder behind an enlarged prostate gland. Diamid and Roy made direct observations of this hypertrophy in the heart by ligating the aorta with a slip-loop ligature and drawing the ligature more or less tight. The heart was connected by means of an appa-

ratus to a recording drum, so that its movements could be distinctly registered. In these observations it was seen that with increased pressure within the ventricle the wall expands in diastole, that is, the heart becomes dilated. But with increase of work thrown upon the heart by tightening the ligature, the fibres do not shorten to the same extent so that all the blood is not expelled in systole. Consequently hypertrophy is always preceded by some degree of dilatation. Hypertrophy is, therefore, always eccentric. There is no such thing as concentric hypertrophy, and the cases so regarded have turned out to be contractions, especially in connection with post-mortem rigidity. So soon as this contraction subsides, it is seen that the cavity of the ventricle is larger than in the normal heart. So long as there is no dilatation there is no regurgitation and no increased work. Consequently there is no hypertrophy. It is only when the muscle begins to flag in its force and fails entirely to expel its contents that dilatation occurs with relative incompetence, and its consequence, eccentric hypertrophy.

Whether the hypertrophy which occurs in these cases is to be ascribed mainly to increase in the size of pre-existing cells or actual increase in the number of cells, is a question which has already been discussed under the general subject of hypertrophy, where the conclusion was reached that the bulk of evidence speaks in favor of the actual increase in the number of cell elements. Adami attributes this increase not so much to a new growth as to a splitting up or division of pre-existing fibres. This author calls attention to the fact that the heart muscle is not a single cell but is the result of the fusion of several cells into an individual unit, so that it is easier for the fibres of the heart muscle to split up into independent territories without derangement of function than in the case of cells composed of isolated units.

In whatever manner accomplished, there is, as stated, no form of hypertrophy so great as that which occurs in valvular disease. The hypertrophy may be confined to certain parts or may affect all parts of the heart. As the hypertrophy is the result of increased work, it begins behind the obstacle and then extends to involve successively other parts of the heart. But the obstacle which is offered is not always immediately behind the valve affected. The increase in size affects the muscle, not from an anatomical but from a physiological standpoint. Thus in the case of the most common form of valve lesion, mitral regurgitation, the first effect is not felt in the left ventricle. On the contrary, the left ventricle may undergo atrophy rather than hypertrophy because it has less work to do. The blood in this case is dammed back into the left auricle, and here is felt the

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first effect of increase of work. The left auricle undergoes some degree of hypertrophy, but the capacity of the auricle in this direction is weak. Blood, therefore, accumulates in the lungs to throw extra work upon the right ventricle. Here, therefore, the first distinct enlargement ensues, and this hypertrophy of the right ventricle, so long as it may still overcome the obstacle in the blood, is said to be compensatory.

But hypertrophy of the heart is not to be looked upon as a mere mechanical process. It is always to be borne in mind that the heart is constructed for the benefit of the tissues of the whole body. The relation is not the other way as it is commonly put. The heart is for the body; the body is not for the heart. Tissues have a way of making known their wants to the heart, and the heart has a way of making known its capacity to the body. These mutual wants are expressed through the nervous system. The heart is stimulated in its action to increased frequency or force by the demand of the tissues. On the other hand, the heart may send out depressing influences, which lower the blood pressure in the distant vessels and thus relieve the work of the heart.

It is a well-known fact that insufficiency in the action of a valve is more common than stenosis, and that the most extreme hypertrophies are found in the face of an insufficiency. It may be readily understood why the heart assumes such enormous dimensions in cases of insufficiency, say at the aortic valves. The extreme hypertrophy in these cases, which makes the heart of man assume something of the proportions of that of the ox (cor bovinum) does not result from the increased stimulus of the impact of blood as it falls back from the aorta through incompetent valves into the left ventricle; the extreme hypertrophy is partly the response of the heart to the urgent demands of the tissues, which are in this lesion so imperfectly supplied with blood, and partly the result of the increased work of the heart staggering with the effort of forcing blood through vessels rigid with arteriosclerosis.

Sometimes the process extends directly from the endocardium to the pericardium. This extension is more likely to be seen in cases of affection of the aortic valve, and Strümpell considers it probable that the excitants of the disease pass directly from these valves through the wall of the vessels to the pericardium to produce the dangerous complication of pericarditis (Fleischer).

Whatever may be the character of the cause, infectious or degenerative, the effect upon the action of the valves is the same. It renders the valves incompetent. The lesion may be of such a character as to prevent the perfect closure of the valves or as to offer obstacle

to the circulation of the blood. When perfect closure is prevented the valves are said to be, *sensu strictu*, incompetent or insufficient. They permit a reflux or regurgitation of the blood. When the valves or the orifices are so affected as to offer obstacle to the circulation of the blood they are said to be stenotic. They constitute the lesions which are called obstructive. Either of these conditions, insufficiency or stenosis, may exist alone. Not infrequently they coincide, that is, the lesion may be of such a character as to make the valves incompetent and at the same time offer obstacle to the circulation of the blood. Such a lesion constitutes a combined insufficiency and obstruction.

Finally, the valves may be incompetent entirely independent of any sign of inflammation or infection. The valves of the heart are reduplications of the endocardium. They are tense, resilient, yielding structures, but the capacity of extension to secure perfect adaptation is limited. The line of apposition of the valves in perfect closure lies at some little distance from their free margins. Therefore any marked increase in size of the orifice makes the valves incompetent entirely to close the orifice. It will be remembered also that the orifice itself changes shape, adjusts the valves, so to speak, at the proper time, to effect a proper closure. Any weakness in the action of the muscles, any dilatation of the chambers of the heart may thus render the valves incompetent. An insufficiency of this kind independent of any manifest disease or change in the endocardium is said to be a relative insufficiency. This relative insufficiency may occur to any valve or set of valves, but does occur most frequently, as will be seen, with the tricuspid valves.

The condition of the various organs under the stasis of valve lesions and the processes of infarction from embolism, are studied in connection with the symptomatology of the disease.

Symptoms.

Sclerotic endocarditis may be entirely without symptoms. Chronic valvular disease from other cause, as from atheroma, may be likewise latent. Where the disease is confined to the wall of the heart it may show no signs. Thus exclusively mural lesions are discovered usually only upon autopsy. Sometimes, however, thrombotic masses from the interior of the heart are washed into the circulation to make known the pre-existence of the affection in embolization of important structures. Some of the cases of sudden aphasia or apoplexy with hemiplegia, certain cases also of embolism of the retina, lungs, spleen, or kidneys, are found to be due to this cause.

As these accidents are rare in this condition, purely mural lesions pass unrecognized.

But even cases of valve affection may escape recognition during life. Where the disease process is insidious and where, under good nutrition, hypertrophy is quickly established and thoroughly sustained, the valves themselves may show no signs. Perhaps it is more true to say that in the absence of apparent or obtrusive signs no suspicion is entertained of the existence of valvular disease. How frequently this condition is the case is best illustrated in the statement of Sir Andrew Clark, so often cited, concerning 684 cases of valvular disease which showed no symptoms which would attract attention to the heart for a period of five years. Further, all cases show exacerbations and remissions. During the remissions there may be periods of quiescence in which an examination not too critical may fail to disclose any lesion of the valves. But even a critical examination at the hands of competent observers may discover no characteristic signs. Loomis reported a case of valvular disease without murmurs; Watney several cases of mitral disease without murmur; Gairdner a case of mitral stenosis without heart murmur; Cryan a case of extreme contraction of the tricuspid and aortic orifice unattended by presystolic murmurs; Bristowe a case of old standing disease of the aortic and mitral valves with hypertrophy and dilatation of the heart, which appeared to have produced no inconvenience until a short time before death. Canstatt reported a case of insidious development of heart disease, hemorrhage from the lungs, and general hydrops shortly before death; on autopsy there was found an absence of a segment of the aortic valve, chalky concrement upon the aortic valves producing stenosis and insufficiency, with dilatation of the left ventricle, hemorrhagic infarction of the lungs. The existence of other disease may completely mask the heart disease. Girandeau found in a number of cachectic patients, who had succumbed to some chronic disease, exuberations on the endocardium which had given rise to practically no symptoms at all during life. The staphylococcus was demonstrated in the lesion in three cases, and in two tuberculous patients this observer demonstrated by inoculation the presence of tubercle bacilli in connection once with the staphylococcus and once with the Bacillus coli. Finally, Vickery reports twelve cases of heart disease unsuspected on account of long latency of symptoms.

Nevertheless, these latent cases are exceptional. Valvular disease reveals itself, as a rule, by distinct signs. The symptoms of chronic valvular disease are general and local. The general signs are to be referred rather to the condition of the myocardium, which, as stated, is always more or less affected in all cases of valve disease. The local

symptoms on the part of the heart itself permit of a diagnosis being made with the greatest accuracy in most cases.

Certainly in by no means a small number of cases the suspicions of the practitioner are excited by the failure in the general health. The first thing that is noticed is the diminution in the vigor. Work which was before undertaken with zest now becomes a drag; the patient is oppressed with monotony. There is a sense of languor and weariness and a tendency to sleep, which, however, is broken by disturbed dreams and from which the patient awakens comparatively unrefreshed. There is at the same time a corresponding abatement in the physical health. The patient feels himself disinclined to effort, becomes easily fatigued. Any unusual effort brings on an attack of palpitation and panting respiration of more than ordinary severity. Rest also does not bring relief so soon. Under these changes life loses its zest. The patient becomes indifferent or irritable.

The origin of chronic valve lesion is so insidious as to escape observation. But nothing is more important than to know as nearly as possible when the disease process begins, as the conservation of the heart depends so much upon protection against exposure and abuse. Patients who may recall a case of rheumatism or other infection which stands in some etiological relation to endocarditis have some advantage over others in whom the process develops without known cause. The attention of most patients is directed to the heart by palpitation and pain. These symptoms depend for the most part upon nervous disturbances and are present often in the entire absence of organic lesion. Nevertheless they do occur in the course of valve lesions, and in these cases a predominant pain refers rather to affection of the aortic, and palpitation to affection of the mitral valves. Dyspnœa also belongs rather to the mitral than aortic valves.

Most of the lesions are mitral, and mitral lesions make themselves manifest sooner or later by disturbance in the circulation in the lungs. This disturbance is preceded by stasis and is attended by changes in nutrition. The stasis is an ectasia, in which process the capillaries bulge and actually take up the space of the air cells. The patient therefore soon becomes short-winded. The condition is noticed first on exercise, in the taking of which the patient pants for breath; in fact, he must often stop to catch his breath. Of course the element of heart failure from coincident myocarditis plays here also a prominent rôle.

The disturbance in nutrition makes itself manifest in the epithelium of the lungs. It probably weakens ciliary action in the bronchial tubes; it certainly interferes with the process of metabolism in

the lungs. The act of breathing is not a mere mechanical process and not a simple diffusion of gases. The interchange of gases, among other phenomena which take place in the lungs, is effected by complex chemical processes which are commonly called vital, and the interference with the circulation not only disturbs the nutrition of the whole body, leading, as stated, to the accumulation of toxins, etc., but also affects the nutrition of the air cells themselves.

Hence it is that an individual affected with heart disease so easily takes cold. The patient takes cold on every exposure, and without any exposure. Every now and then he suffers from some catarrhal process which distinguishes itself not only by its frequency, but by its obstinacy. The suspicion of incipient tuberculosis is excited, and is further strengthened by the pallor and progressive marasmus of these cases. The cough, the expectoration, the hæmoptysis which sometimes occurs, seem to furnish conclusive evidence, and tuberculosis is only excluded at times by the negative results of repeated examinations of the sputum. It must not be forgotten that tuberculosis may develop at any time under the defective nutrition of heart disease, but it may be remembered, as stated, that the conditions of hyperæmia which characterize most cases is but little favorable to the development of the tubercle bacillus. It is noticed, however, that there is no fever, the presence of which is characteristic of tuberculosis. The diagnosis in these cases is sometimes satisfactorily established, it may be said here, *ex adjuvantibus*. It is seen that the catarrhal processes, dyspnœa, etc., disappear quickly under the use of digitalis, especially when it is combined with a little ipecac. Then tuberculin gives no reaction in heart disease.

It is seen soon that the blood-making process is at fault. The face loses the hue of health, becomes pale, is sometimes puffed; the mucous membranes are not so red; the whole body is not so warm; the feet easily become cold. Sometimes there are chilly sensations with attacks of headache, conditions which excite the suspicion of nephritis, the coexistence of which is not infrequently actually established. Interrogation brings out the fact that the individual has been subject to rheumatism or has suffered from some of the numerous infections of sepsis, perhaps many months, even years, before the present attack. There may be as yet no obtrusive physical signs to point directly to the seat of the disease, so that the physician comes upon the lesion in the valves in the course of the examination of the whole body.

The French writers describe a physiognomy which they consider indicative of the various forms of heart lesion. The mitral facies is described as venous, congested, and blue, while the aortic is more

distinctly anæmic and pallid. This physiognomy is seen more especially in connection with the mitral valves in stenosis of the auriculo-ventricular orifice and is present in insufficiency only in advanced cases or in those cases in which the insufficiency is complicated by stenosis.

Pallor may coincide with cyanosis in mitral stenosis. Cyanosis in conjunction with the icteric tint which is sometimes seen produces a peculiar characteristic, almost greenish tinge. Icterus itself sometimes becomes extreme. Icterus occurs in connection with gastro-duodenal catarrh and stasis in the liver, and may result in extreme cases from absorption of coloring matter from the lungs in hemorrhagic infarction.

In another class of cases the patient complains rather of the lungs than of the heart. There is cough, sometimes with hæmoptysis. There may be chilly sensations with sweats, and these things taken in connection with the loss of weight and anæmia direct attention rather to tuberculosis of the lungs.

In still another class of cases the patient directs the attention of the physician more especially to the stomach on account of dyspeptic signs. Sometimes the symptoms on the part of the stomach assume prominence. Sometimes, but rather in the advanced cases, the anorexia is profound and there is an absolute and unconquerable aversion to every kind of food. In other cases attention is called to the kidneys on account more especially of the excess of urea with the deposit of urates, or to the brain on account of vertigo or headache. As already stated, the embolic processes may be the first symptoms, and patients have actually consulted physicians for the relief of eruptions upon the skin produced in this way.

In a certain class of cases, further, the patient consults the physician on account of œdematous effusions about the ankles, complaining of the feet, that the shoes are tight, especially at night, while the condition is not to be seen in the morning.

Lastly, a large number consult the physician to be relieved of symptoms on the part of the heart itself. The patient complains of early exhaustion, palpitation, dyspnœa, etc. It is true, however, as stated elsewhere, that those who complain most of the heart seldom suffer anything more than nervous disturbance. Except in aggravated forms or in the last stages of the disease the subjective symptoms rarely point directly to valvular disease.

The pulse which is instinctively examined first furnishes information of decided value. So long as compensation is perfect the pulse may show nothing unusual or abnormal; but as compensation is never absolute, there are times in which the pulse offers, even in

favorable cases, at least corroborative evidence. When the compensation begins to give way the pulse becomes irregular, shows arhythmia, or in still later stages flutters under delirium cordis. Thus in mitral regurgitation, which is the most frequent lesion of the heart, the pulse may show no alteration for a long time, even for years. When a change is seen it is noticed that the pulse, while perfectly regular, is rather small and weak, for the reason that the left ventricle loses part of its blood by regurgitation, and the aorta and great vessels receive a diminished blood supply. This condition is even more marked in a case of mitral stenosis, as the quantity of blood received by the left ventricle is still less, more of it being retained in the left auricle at the time of the beginning of the systole. In stenosis of the aortic valve, the pulse is small but hard and incompressible, because only a small quantity of blood is expressed under the great force of the hypertrophied ventricle through the contracted orifice. The pulse in these cases is said to be wiry. Most striking is the pulse of insufficiency of the aortic valve. In this case the blood is propelled with great force by the extreme hypertrophy of the left ventricle, only, however, to fall back again suddenly through the incompetent valves. Thus the arteries are subjected to sudden distention and equally sudden collapse.

Geigel reported a case of mitral insufficiency in a girl aged twenty, affected with erysipelas, who showed under light psychical excitement the pulsus bigeminus. Auscultation of the heart showed that the second contraction was always marked by a systolic murmur at the apex and accentuation of the pulmonary tone, while the first systole was perfectly normal. Geigel very properly considered it a functional insufficiency due to weakness of the papillary muscles.

Popoff observed the pulsus differens in several cases of mitral stenosis and insufficiency. As this symptom was observed in cases in which the autopsy showed none of the usual causes of the pulsus differens (aneurysm of the aorta, contraction of the subclavian artery by arteriosclerosis, tumors, etc.), and as the smaller and weaker pulse was always found in the left radial artery, Popoff ascribed it to a compression of the left subclavian artery by the dilated left auricle and distended pulmonary artery. Some support for this view is found in the fact that the pulsus differens is only encountered in cases where the heart's action is insufficient.

Arhythmia indicates no exact condition of disease. In fact, arhythmia is often found in perfect health. This was known as long ago as in Galen's time, and Prosper Alpianus and de Haën were familiar with the fact that arhythmia may occur when there is no change of the heart and no reflex influence that can be discovered.

The alternations in the volume of the pulse, at times small and at times large, correspond, according to Nothnagel, with alternations in the action of the heart.

A venous pulse is sometimes to be seen in the neck. The great veins in the neck may show a presystolic swelling in the physiological state, while the pathological venous pulse shows its chief swelling in connection with the systole of the heart, constituting the true venous pulse which is characteristic of tricuspid insufficiency.

The blood itself seems to be but little affected by valve lesion. The specific gravity is usually normal or is but little reduced (Hammerschlag, Schmalz) and the proportion of hæmoglobin is nearly normal (Bamberger). It is only when compensation has become greatly disturbed, especially in the presence of relapses of endocarditis attended by fever, that alterations in the blood, leucocytosis, poikilocythæmia, may be observed. Oertel finds the blood hydræmic, Bamberger finds it inspissated in the stage of broken compensation and in degree according to the amount of venous stasis.

The physical signs are much more positive, though a certain training in physical examination is necessary to elicit them and appreciate them properly.

Examination of Valve Lesions of the Heart.

The examination is made best with the upper half of the body entirely disrobed. Such examinations apply of course only to the male, and it is partly on this account and partly for other obvious reasons (mammary glands, panniculus adiposus, etc.) that the study of heart disease is, as a rule, so much more satisfactory in the male sex. It was the reserve imposed by the sex in a patient of considerable *embonpoint* which led Laennec to roll up a piece of gold-beater's skin in the form of a tube and interpose it between the ear and the chest. This was the birth of the stethoscope which practically laid open the interior of the heart and illuminated its deepest recesses. The patient should stand opposite a window, facing a good light, or if too ill should lie semirecumbent in bed with the shoulders well raised.

Inspection sometimes reveals at a glance general dropsy or a local œdema, especially of the lower extremities. Sometimes a light cyanosis of the face betrays the character of the disease. In bad cases of exhausted heart the strained efforts of respiration marked by the action of the auxiliary muscles, by the play of the alæ nasi, indicate the weakness of the heart, which is also depicted in the pallor and anxiety of the face. The thorax in the region of the heart may be distended. This vaulting of the chest or *voussure* is to be observed

only in the case of the young where the thorax is still resilient. No amount of hypertrophy may bulge the chest of an adult. Moreover, the condition is not frequently observed even in childhood, and when present is rather indicative of pericardial effusion than hypertrophy of the heart. The eye of the examiner searches the chest for the situation of the apex stroke. The location of the apex conveys valuable information. Any dislocation of the apex is noticed at once as the fixed point from which to define the outlines of the heart. Under the hypertrophy which the heart undergoes in chronic valve disease, the apex is usually dislocated downward and to the left, sometimes as far to the left as the axillary line. Sometimes the stroke of the heart is unusually light; sometimes it is entirely invisible. On the other hand, it may be too strong so that the whole precordial region, the whole thorax, even the whole body, may be visibly agitated by the powerful action of the heart. Inspection reveals also the distention and throbbing of vessels in the neck, or the sudden collapse which occurs after distention in the arteries and veins.

Palpation perceives these changes with even greater nicety. A vaulting of the chest may be felt when it may not be seen and the apex stroke may be perceived by the touch when it may not be apparent to the sight. Moreover, the hand applied to the chest may better appreciate the impact of the whole heart. It was Corvisart who first of all felt the *frémissement cataire*, the vibratory thrill which is imparted to the fingers by the friction of blood over roughened surfaces. The vibratory thrill is felt most frequently in obstructive lesions, though it is sometimes found in connection with insufficiency, especially of the mitral valve. The location of this thrill, whether at the base or the apex, is to be distinctly noticed, with the time of its occurrence with relation to the phases of the action of the heart.

Percussion furnishes even more valuable signs. Under percussion the outlines of the heart may be fixed with a certain degree of accuracy. It is noticed that the dulness extends as high in the chest as the second or third rib and reaches over to the right beyond the right border of the sternum. The outlines of dulness, both relative and absolute, may be traced with colored crayon and subsequent changes in the diameters may be followed up in this way.

The determination of the exact outlines of the heart by percussion is difficult and is often impossible. Distinction has hitherto been made between the region of absolute dulness, which represents the part of the heart directly apposed to the wall of the chest, and relative dulness which represents the part of the heart which is covered by a more or less thick layer of lung tissue. Piorry distinguished the

relative dulness as extending one and one-half to two inches above, and one and one-half to one inch about the region of direct application of the heart to the wall of the chest. Williams speaks of the shading off of the heart dulness under the lung tissue. Meyer from his anatomical studies found the greatest difficulty in fixing the *right* border of the heart. Bamberger established it at the right border of the sternum. Gerhardt fixed it as an irregular line beyond the left border of the sternum but never even reaching the middle, to say nothing of the right border. Walshe fixes it in the middle of the sternum from the fourth to the sixth rib. Friedreich declares that it is only in exceptional cases that it reaches the right border of the sternum. Most writers admit that the sound is propagated by the vibrations, by the peculiar resonance of the sternum itself. Ebstein, who used the percussion palpation method, found the resistance of the heart to extend beyond the right border of the sternum. Ewald finds the right border in a curved line, the upper end of which reaches to the cartilage of the second rib, the lower end to that of the fifth rib, while the bottom of the arch, whose concavity is directed toward the right border of the sternum, lies in the third intercostal space.

The same difference of opinion is held regarding the upper border of dulness. Conradi fixes it in the third intercostal space, Meyer, Stümpell, and Kobelt declare that it reaches to the second intercostal space.

So the breadth of the heart dulness is variously estimated. Strümpell fixes the maximum breadth in the fourth intercostal space at 7 cm., Kobelt at $10\frac{3}{4}$ cm., Reese at 11 cm., Schlaefke at 15 to 17 cm. Meyer declares that the dulness in the third interspace varies from $2\frac{1}{2}$ to $6\frac{1}{4}$ cm. These variations are so great as to have led Skoda to establish no exact dimensions. Nothing shows the differences of opinion better than the statement of Conradi, who declared that in many cases he could find no trace of heart dulness, as the heart was everywhere covered by the lungs. Skoda says that the sound in the region of the heart is never perfectly dull. Matterstock found the heart dulness in the same individual to vary at different times of the day.

The greatest difficulty really pertains to the fixation of the left border of the heart, which is least constant of all, as it is so variously overlapped by the lingula of the left upper lobe of the lung and by the distended stomach with its solid, fluid, or gaseous contents. The best illustration of the variation of the volume of the heart or the various estimates of its volume is given by Heitler, from whose exhaustive study of this subject most of these points are taken, in the cases of several patients who had visited a number of the distin-

guished clinicians of Europe, some of whom maintained that there was hypertrophy, while others declared equally positively that the heart was perfectly normal in its dimensions.

For all practical purposes, therefore, so far as percussion is concerned, the heart may be considered enlarged only when the dulness extends or begins upward above the third rib, to the right when it reaches to or beyond the right border of the sternum, and to the left along the line of the apex, whose situation may be accurately determined, to the left of the mammillary line. There is no lower border, as the dulness of the heart passes directly over into that of the left lobe of the liver or into the sounds which belong to the stomach.

Auscultation furnishes extremely valuable evidence in the alterations of the sounds of the heart. The contributions of auscultation were so lavish at first as to have led to an overestimate of this means of diagnosis; so that in the reaction which followed there was a tendency to depreciate the value of the information thus obtained. It is sometimes said even now that of all the means of diagnosis auscultation furnishes the least positive and the least reliable evidence. This is not true, however, and while it is possible to be misled for a time, especially by the so-called inorganic or accidental murmurs, the estimate which the clinician puts upon auscultation is indicated by his immediate use of it, brushing away, as it were, all other means at first in his instinctive impulse or eagerness to apply the ear to the chest to listen and let the heart speak for itself.

The natural sounds of the heart are produced partly by muscular contraction, partly by the collision of blood, and other causes, apex stroke, etc., but chiefly by the closure of the valves. As the sounds at the base of the heart are produced almost entirely by the action of the valves, anomalies revealed by auscultation are here of more positive value than in the case of sounds at the apex. All natural sounds of the heart are very characteristic. The first sound is comparatively long and sustained, while the second sound is short and abrupt. In his search for imitation, the clinician may not refuse to recognize the nearness of the sounds *lubb, dupp*. After the second sound is the rest. This sequence, the long sound, the short sound, and the rest, in regular order, constitutes the rhythm of the heart.

Disease of the valves, that is, lesions of the valves or of the orifices which the valves close, change the character of the sounds in every conceivable way. Lesions may exist, as stated, and still not greatly interfere with the action of the valves. In such cases the natural sound may be but little or not at all affected. On the other hand, extensive lesions, as the rupture of a valve or the actual severance of a segment from its base, entirely destroy the natural sound and

substitute something totally different. So the anomalous sounds vary in every degree of intensity and character. Frequently the sound is soft and blowing, like a whispered "*who*." Such a sound is said to constitute a blowing murmur, it is heard oftenest in muscular insufficiencies. Sometimes the sound is creaking or grating like the crunching of snow or like the creak of leather, etc. Sometimes the sound strikes a musical note.

Murmurs vary also in intensity and duration. Sometimes they are so faint that they may be discovered only by a trained ear, sometimes they are so loud as to be appreciated by every one, by the patient himself, or even at some distance from the body. Sometimes they last for a period that is almost inappreciably short, sometimes they are prolonged over a considerable period. Where tendons are ruptured or valves are split or torn from the base, the sound may be prolonged to assume something of the character of a scream. This is the *bruit piaulement* of the French. In still other cases they may be present only at certain times; thus exercise of the body in stimulating the heart may make audible sounds which were too faint to be heard before. On the other hand, in the presence of tumultuous irregular action of the heart, all the sounds may be confused and indistinct and may be rendered clear only when the heart muscle is toned to regular action by rest or by some heart stimulant. Finally, it must be remembered always that all sounds become more or less inaudible as the heart's action becomes more and more weak, so that sounds which may have been distinct for a long time may disappear entirely *sub finem vitæ*.

As these sounds or murmurs are produced by lesions of the valves or orifices, they correspond in time to phases of the heart's action. In this regard estimate is taken only of the ventricle, and the sound is said to correspond with the contraction or dilatation of the ventricles. Thus the sound is heard in connection with the systole or diastole of the ventricle.

Sometimes, as stated, the sound actually substitutes the natural sound. In other cases it may be appended to it, or in still other cases it may precede it. So sounds are said to be systolic, subsystolic, and presystolic in time. The same limitations apply, though with less frequency, to the diastole.

Under anything like a regular action of the heart it is usually easy to determine whether a murmur belongs to the systole or diastole, and any doubt may be dispelled, as a rule, by watching the pulsation of the carotids in the neck, by applying the fingers to the pulse during auscultation of the heart, or by feeling the apex stroke of the heart. But when the action of the heart is irregular, tumultuous, and

violent, or when on the other hand it is feeble and fluttering, and the pulse shows corresponding irregularities or vibrates like a loose string, or when for any reason the apex stroke may not be felt, it is at times difficult or impossible to differentiate the diastole. But it is at all times important to discover the existence of a diastolic murmur, or whether any faint murmur that may be heard belongs wholly to the systole or partly to the diastole. Sometimes this question can be settled by appreciation of the periods of time occupied by the revolutions of the heart. Thus investigations have shown that under normal conditions with a diminished frequency of pulse (60) about thirty per cent., with an increased pulse (100) three per cent. of the whole revolution of the heart belongs to the systole, and that the true diastole in reduced pulse occupies three-fourths to four-fifths, on the average about one-half of a second, and with a higher pulse always one-third of a second.

Murmurs vary, as stated, in every degree of intensity. They vary also in the range in which they may be heard. Sometimes murmurs are quite strictly circumscribed about the region of the valves or orifices at which they occur. Sometimes they are widely disseminated from the site of their origin, and they may usually be traced into the great vessels which issue from the heart. Thus a murmur developed at the orifice of the aorta may be heard in the carotid arteries.

Griffiths describes cases of wide dissemination of sound over the chest, and Federici, Vanni, and Petrazzini of still wider over the body. Oddo reports a case of mitral regurgitation and stenosis in which the murmur could be heard not only over the whole thorax but also over the skull, face, along the entire vertebral column, over the arm, over the pelvis, and over the leg in varying intensity down to the lower third of the tibia. Federici believed that the murmur in these cases was taken up by the sternum and propagated over the skeleton, as it was always less intense over the soft parts. Vanni held that the sound was always carried through the vessels as he could stop it by ligating a limb. Oddo accepts both explanations.

In all cases the localization of a sound does not depend so much upon the range or dissemination as upon the point at which it is heard in greatest intensity. For instance, the sound produced by a mitral insufficiency *may be* heard all over the heart or even in the back between the shoulders, but it *is* heard in the greatest intensity at the apex. Thus a sound which may be appreciated anywhere over the chest, in front or behind, may be traced to the point of greatest intensity and so the source of the sound may be fixed. These are two cardinal points in the localization of valve lesion. With the dis-

covery of a bruit, therefore, the practitioner asks himself at once where it is heard in the greatest intensity and synchronous with which sound of the heart.

In the study of valve lesions the whole right side of the heart may for all practical purposes be discarded, at least at first, as lesions of the valves on the right side of the heart are very rare. The attention of the practitioner is directed then to the study of the mitral and aortic valves and their orifices. Lesions of the mitral valve are heard in the greatest intensity at the apex, but much depends here upon the position of the patient. In the upright or semi-recumbent posture, or more especially when the body is bent forward, the apex strikes forcibly against the wall of the chest and the mitral valve is in this situation nearest to the ear of the auscultator. But if the patient lies flat upon his back the heart falls away from the thorax and the mitral valve may be actually nearer to the auscultator at the base of the heart. The sounds of the aortic valve are best appreciated at the base of the heart at the junction of the cartilage of the second rib with the sternum on the right side. The nearest point to the ear lies a little behind the sternum, where the distance from it in the adult amounts to two centimetres. Sometimes a murmur may be elicited only by change of posture. Azoulay recommends the adoption of a particular posture to raise the blood pressure in the heart and thus intensify any lesion that may be present. The patient lies with his head high, raises his arms at right angles to the body and flexes the legs upon the surface of the abdomen. In this posture the action of the heart is retarded and any murmurs present are distinctly intensified. Azoulay calls this position *l'épreuve des valvules* (the valve test). This posture should be adopted with caution, however, as it may be dangerous in the presence of any great weakness of the heart, in any pronounced arteriosclerosis, or in the presence of an aneurysm of the aorta. The posture is especially valuable in the diagnosis of complicated valve lesions, as of the mitral and aortic valves, and for the recognition of the galop murmur.

Now, while it is true that lesions of the right heart are rare, it is also true that certain alterations which change the size of the right auriculo-ventricular orifice are common, so that relative insufficiency of the tricuspid valve is a quite frequent condition. Such a lesion indicates dilatation and must be recognized in order to prove the true gravity of the case. Moreover, while direct lesions of the pulmonary valve are so rare as to be almost never encountered, the sound of closure may be intensified on account of increase of blood pressure, so that the situation of the sounds of the tricuspid and pulmonary valves must be distinctly determined. Lesions of the tricuspid valve

are heard best to the right of the sternum at the junction of the cartilage of the sixth rib. As these lesions are most frequently relative and are developed in consequence of dilatation, the position of the valve becomes somewhat changed, so that the sound of relative insufficiency is usually heard in greatest intensity as low as the end of the ensiform cartilage, where the pulsation of the ventricle may be distinctly seen.

The pulmonic valve sound is heard best at the base of the heart at the junction of the cartilage of the second rib with the sternum. As the sound may not be propagated so readily through the intervening bone, alterations of sound connected with the aortic and pulmonary valves are actually appreciated in greatest intensity in the second intercostal space to the right and left of the sternum respectively. The pulmonic valve really gets nearest the surface just behind the cartilage of the third rib.

Alterations in the character of the sound are usually most characteristic, but alterations in intensity may furnish information of great value. This is especially the case in connection with the pulmonary valve sounds, the accentuation of which is perhaps the most valuable single factor in the diagnosis of insufficiency of the mitral valves. As a general rule it may be said that the first sound as appreciated at the apex is three times as strong as the same sound heard at the base. The weakest of all the heart sounds is that which attends the opening of the aortic and pulmonary valves under the efflux of blood from the ventricles. This sound is practically inaudible. The second sound at the base is caused by the abrupt closure of the pulmonary and aortic valves. Of these valves the pulmonary closes with the loudest tone in about the proportion of six to five. When therefore it is proposed to estimate the accentuation of the second pulmonary sound, regard should be had to the second aortic sound for comparison. "When the second aortic sound is relatively weak a normal intensity of the pulmonary sound would appear relatively strong" (Vierordt).

Ewart maintains that the second pulmonary may be always distinguished from the second aortic sound by the fact that the pulmonary sound may be heard only in a very circumscribed region, namely, at the second left interspace near the sternum, while the aortic sound may be heard over a wide area, even at the apex. Frozen sections show that the pulmonary valves are so superficial as to be unable to disseminate much vibration, while the aortic valves lie close to the septum and thus agitate the whole heart.

As already stated, lesions of the valves or the orifices may be so disposed as to interfere with the escape of blood from the cavities of the heart or to permit the reflux of blood into the chamber, whence it has already been discharged. Lesions of the valves are said to be,

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therefore, of two kinds, obstructive and regurgitative. Synonyms for these expressions are stenosis and insufficiency. A relative insufficiency will develop a murmur as distinct as that from an absolute lesion. Relative insufficiencies are observed most frequently at the mitral and tricuspid valves on account of impairment of muscular tone or actual disease or degeneration of muscle substance.

Given now these various factors—the existence of a murmur, the point at which the murmur is heard in greatest intensity, and the time when it occurs, whether synchronous with the systole or diastole—there is required only the student's knowledge of the course of the blood through the heart to establish the exact nature of the lesion; that is, to fix upon the valve affected and to specify the character of the affection, whether it be an obstruction or a regurgitation. Thus a murmur heard in greatest intensity at the apex of the heart and synchronous with the first sound is developed in consequence of insufficiency of the mitral valves, because the contraction of the ventricle which occurs at this time, and which should drive all the blood into the aorta, expels part of it through incompetent mitral valves into the left auricle whence it came. The murmur which marks the insufficient closure is heard in the greatest intensity at the apex, because it concerns the mitral valve and is synchronous with the first sound of the heart, which is produced partly by the contraction of the ventricle.

Anomalies of sound are also produced by irregular vibrations from collisions of opposing currents of blood and by friction of the blood current as it is forced through contracted or partially occluded orifices. The sound varies in character and intensity according to the character of the lesion. It is rarely the case that a valve is affected in such a way as to show either insufficiency or obstruction alone. The conditions are much more frequently conjoined, that is, the lesion is of such a character as to permit regurgitation while at the same time it offers obstacle to the passage of the blood. Such a lesion is said to be double; there is both insufficiency and obstruction and the murmur is double. The same rules apply in deciphering the double murmur—that is, attention is paid to the point at which each murmur is heard with the greatest intensity and to the time with reference to the movements of the heart. Thus a double murmur heard in greatest intensity at the apex would indicate insufficiency as well as obstruction at the mitral orifice. As a rule, the obstruction is slight compared with the insufficiency, so that the presystolic or diastolic is much less intense than the systolic murmur. Where the obstructive murmur is so slight as to be almost imperceptible, or perhaps to be excited only under increased activity of the heart, it is, as a rule,

practically disregarded. When, however, this murmur is at all pronounced it assumes prominence and gives much greater gravity to the case, for in a general way it may be said that a diastolic or presystolic murmur speaks more absolutely for organic lesion than a mere systolic murmur, which may in this region represent simply a relative insufficiency.

When two murmurs are present it may be ascertained that one is heard in the greatest intensity at the apex and the other at the base. Here, too, the character of the lesion is disclosed by the observance of the same rules. Thus the murmur heard in greatest intensity at the apex and synchronous with the first sound of the heart would indicate a mitral insufficiency, while a murmur heard in the greatest intensity at the base to the right of the sternum, also synchronous with the first sound of the heart, would indicate an obstruction at the aortic orifice. When the two murmurs respectively heard, one in the greatest intensity at the apex, the other at the base, correspond with the second sound or diastole, the murmur at the apex indicates a stenosis at the mitral orifice, while that at the base indicates insufficiency of the aortic valve. Should it happen that the murmurs differ in time, the one at the apex being synchronous with the first sound of the heart while that at the base is synchronous with the second sound of the heart, the murmur at the apex would indicate insufficiency of the mitral valve, while the murmur at the base would indicate insufficiency of the aortic valves.

Thus the mere knowledge of the course of the blood in its circulation through the heart enables the observer to fix upon the valve which is affected, and not only that, but to determine the character of the interference. As will be seen in the study of the prognosis, it is by no means a matter of indifference which valve of the heart is affected or in which way it is affected, for though the main factor is in all cases the condition of the heart muscle itself, the character of the valve lesion has much to do with the degree and time of occurrence of the degenerative change.

Secondary Symptoms.

There remain now to be considered the more remote effects of interference with the circulation in the heart. These effects may be summed up under three heads:

(1) Those which arise from defective blood supply;

(2) Those which depend upon stasis;

(3) Those which result from embolization of distant vessels.

As stated elsewhere, the brain is the most sensitive of all organs,

hence it feels at once any defect or interference with the blood supply. Brain defects are felt most frequently and severely in connection with contraction at the aortic orifice, that is, in obstructive lesions in which, notwithstanding the hypertrophy of the left ventricle, but a small quantity of blood is forced under great pressure through the contracted orifice. Where the lesion is at all pronounced or in any case where the heart muscle begins to flag in its force, the patient suffers attacks of faintness, vertigo, and syncope. It is easy to understand that exercise of any kind increases the difficulty, so that the patient is at this stage compelled to reduce his efforts or frequently to take rest. All the organs of the body suffer the same ischæmia. Along with the reduction in the propulsive power of the heart and loss of resilience in the arteries on account of imperfect filling of the vessels, there is a tendency to accumulation in the capillaries and in the venous system, so that while the various intestinal organs, especially the liver and kidneys, may show for a time the condition of anæmia, this state is soon substituted by the hyperæmia of stasis. In all cases, however, the various organs suffer from lack of nutrition and as a result of this change various toxic products accumulate in the blood. That the defective oxygenation leads to an increased consumption of the albuminoid matters of the blood is shown by the increased excretion of uric acid, which in the presence of more oxygen would be oxidized into urea. Thus Fleischer found in two cases of extreme heart weakness in consequence of valve lesions, 1.3 and 1.8 uric acid excreted during the day; that is, six times the normal amount.

The effects of stasis are noticed more frequently when the valves are incompetent or at least they are observed so soon as the compensatory hypertrophy begins to give way. Thus in insufficiency of the mitral orifice, which is the most frequent lesion, the blood accumulates in the left auricle and, so soon as the auricle becomes distended, in the pulmonary veins and in the lungs. Stasis in the lungs makes itself manifest by frequency of respiration, by attacks of dyspnœa, at first under effort and afterward spontaneously, also by disturbed nutrition, by the processes of catarrh; finally, in extreme cases, by cyanosis and hæmoptysis.

The first effect of insufficient filling of the arteries is marked by pallor of the skin. When the quantity of blood is lessened the arteries are correspondingly reduced in size, and this contraction of the blood-vessels leads to pallor.

Dropsy.—When the capillaries in the lungs are kept in a state of distention the blood is dammed back into the right ventricle and thence into the right auricle, which soon becomes unable to unload itself.

Hereupon ensues a condition of stasis in the systemic veins. The continued or increasing distention of the veins and insufficient nutrition of the walls of the veins lead to transudation of their more fluid contents. Thus œdema develops about the ankles, being observed at first only at night after the work of the day, later, as the fluid accumulates, continuously. At this time the skin upon the dorsum of the foot or about the ankles pits on pressure, or where the shoe is tight the fluid accumulates above its upper border and the œdema subsides or becomes more diffuse when the shoe is removed. Still later the fluid mounts the legs. It is observed in lighter degree first in the anterior tibial region because of the hard substratum of the shin bone. In fact, it is common practice to test the skin in this region first. The skin is said to "pit" upon pressure. Any continuous pressure upon the leg, as by a garter, leaves a circular depression. The subcutaneous œdema continues to ascend to show itself upon the thighs. Next the genital organs are involved. The labia are distended, the scrotum may be converted into a large, translucent globe, the prepuce is swollen and convoluted so as even to offer obstacle at times to the discharge of urine. The dropsy mounts to the tissue of the abdomen and invades the sac of the peritoneum.

Dropsy now rapidly extends over the whole body, constituting anasarca. The distention may be so great as to stretch the skin, which looks shiny or glazed, and may be cold, livid, or blue. The defective nutrition from compression and from the circulation of toxic elements in the blood leads often to a peculiar hyperplasia of the subcutaneous connective tissue, so that the skin itself is thickened upon the enormously distended legs. The hyperplasia has been ascribed to a lymphangitis and the condition has been considered and described as a kind of elephantiasis. This same process is not infrequently observed after tapping, and under it the thickened skin may be bound down to the subcutaneous tissue. The skin now no longer glides over the subjacent structures, but is agglutinated with them to form a solid and inseparable mass. Sometimes the skin ruptures to permit the escape of clear serum, which trickles down in streams and discharges in the course of time great quantities of fluid. Dermatitis, erysipelas, or gangrenous processes may now develop in these regions. The accumulation may be more marked in one extremity, as for instance in one hand or foot. The skin of the back of the hand or of the dorsum of the foot is puffed up and distended and has a translucent look. These localized processes are due to thrombotic occlusions, usually in the brachial or femoral veins. In the upper extremities, the left is attacked by preference because of the obliquity of the left brachio-cephalic vein.

As the abdominal muscles themselves are loaded down and the excursions of the diaphragm are limited by the accumulation in the peritoneum, respiration becomes more and more difficult. Finally the pericardium fills and the action of the heart is interfered with, in extreme cases arrested, and the patient is commonly said "to be drowned in his own fluids." But more frequently the interference with the action of the heart conjoined with the great weakness of the heart muscle lead to œdema of the lungs of more or less sudden development, which is manifested by cyanosis, dyspnœa, suffocation. The acute œdema of the lungs, which announces itself by the extreme dyspnœa of cardiac asthma with the expectoration of abundant albuminous fluid often tinged with blood, has been variously attributed: by Grossmann to a poisoning of the nervous system, like that which occurs after the administration of muscarin, which excites a spasmodic contraction of the left ventricle; by Walshe, to paralysis of the left ventricle, and by Bouveret to a vaso-motor œdema such as that which occurs in the course of Bright's disease.

Dropsy is usually accounted for on the mechanical theory of obstruction. This theory seems to have been established by the experiments of Lower (1860), who showed that ligation of a great venous trunk is followed by dropsy in the parts behind it. Bouillaud established the fact that most œdemas are due to obliteration or obstruction. Thus obstruction of a principal vein of a member is followed by an œdema confined to this member. Obstruction of the heart is followed by œdema of the whole body.

But these statements have not gone without controversy. Hodgson (1815) and Reynaud (1820) published cases of obliteration of the femoral vein which were not followed by infiltration of the lower extremities. Rannier (1869) ligated the two jugular veins but observed no œdema in consequence. This author made the same observation after ligation of the femoral vein and the inferior vena cava. In a second series of experiments, after having tied the inferior vena cava, he cut the sciatic nerve on one side. Twenty hours afterward there was observed enormous œdema of the member on that side. Some nervous influence would seem, therefore, necessary to the production of œdema. Vulpian, Strauss, and Picot ascribed to the nervous system a preponderating influence in the development of œdema. There is no doubt that two phases may be observed, one the physical due to occlusion of the vessel, the other the chemical due to the changes which take place in the blood and in the walls of the blood-vessels (Petit).

The impeded escape of blood from the brain gives rise to hebetude, confusion of ideas, headache, and in the very last stage of fail-

ing heart power, to delirium and coma. Extreme stasis leads finally to œdema of the brain.

When the tricuspid valves have become incompetent and the jugular veins dilated beyond the capacity of the valve at the bulb, the veins are seen to throb in the neck with every pulsation of the right ventricle. The great veins are swelled under stasis and the cervical veins become especially prominent. As the emptying of the veins into the thorax is impeded by the act of expiration, it is noticed that the cervical veins especially are more dilated at this time. When the valves of the vein no longer suffice to control the calibre, that is, when they become relatively insufficient on account of dilatation of the veins, the pulsation of the right auricle is propagated through the veins of the neck as an undulatory contraction synchronous with the movements of the heart. The patient may now no longer remain in the recumbent posture but must assume the semi-recumbent or sitting posture, in order to secure the fullest emptying of the veins and the most ready assistance of the auxiliary muscles of respiration.

Cardiac Cirrhosis.—The liver is peculiarly sensitive to stasis because of the double set of capillaries in the vascular system. The portal vein begins in a system of capillaries which converge to constitute a very short trunk, and the trunk of the portal vein breaks up into an extensive system of capillaries in the substance of the liver. The blood from these capillaries is collected again into the radicles of the hepatic veins. Any stasis of blood in the liver leads, therefore, to enormous distention, so that the liver may be double the natural size. Sometimes in cases of extreme stasis the edge of the liver is felt on a line with the umbilicus. The disturbance of nutrition which ensues in consequence of protracted stasis leads to hyperplasia of the connective tissue, which in its contraction displays the peculiar induration which is distinguished from that produced by the irritant effect of alcohol, as the "cardiac" cirrhosis. Hutinel found a genuine interstitial proliferation of the connective tissue with all its consequences in the liver of children affected with valve disease. Hanot, who considers the condition frequent, employs the rather confusing term "*asystolie hépatique*" to express the relation between the heart and liver, and declares that the signs on the part of the liver may overshadow all other signs. Alcoholism, infectious disease, auto-intoxications favor the process.

Stasis in the liver is indicated by interference with the function of the organ. The liver protects the body against auto-intoxication, and this antitoxic is one of its chief functions. Poisons are introduced into the intestine with the foods (alkaloids, potash salts, alcohol, etc.), or as products of the action of micro-organisms, pto-

maines, toxins, toxalbumins. Material is brought also by the blood for the formation of bile, urea, etc. The liver in health disinfects and purifies all these matters. Slight interference with this action produces the discomfort and distress of "biliousness"; abolition of action produces the ominous signs of icterus gravis.

Where the tricuspid valve has become relatively incompetent, the same distention of the veins observed in the neck may be seen also in the liver; or if the pulsation of the liver may not be seen, it may be felt by the hands applied over the liver at the side and front of the body.

In all cases where the circulation in the liver has been brought to the condition of stasis, retrograde changes soon set in, marked especially by fatty degeneration and the alternate arrangement of the black spots, which represent an extreme stasis in the radicles of the hepatic veins, with the yellowish tint of fatty degeneration and the reddish hue of disorganized blood, give the liver upon section the mottled appearance which is known as the "nutmeg" liver. The nutmeg liver is recognized at a glance by pathologists as an expression of extreme stasis from incompetency of the heart muscle.

The stasis extends so as to involve also the radicles of the portal vein and of the mesenteric veins in the gastro-intestinal tract. This venous hyperæmia soon develops in the stomach dyspepsia with its train of symptoms, anorexia, arrest of secretion, gastrectasia, etc. The distress of defective digestion is so often referred to the heart as to have received the significant name of "heart burn." In the intestine there is chronic catarrh, alteration of secretion, constipation, hemorrhoids, etc.

Harley called attention to the danger of sudden death from distention of the stomach, in connection with the disturbance of digestion in heart disease. In certain cases the symptoms on the part of the gastro-intestinal canal may be ascribed to œdema of the mucous membrane, in other cases to the alteration of metabolism, and in still other cases to uræmia.

Somewhat similar changes occur in the *kidney*. The whole organ is enlarged. The veins are full to distention, the urinary tubules are compressed, the secreting structure is altered, the quantity of urine is greatly reduced, and what is passed is heavy, turbid, and dark. The fact is, the quantity of urine and the character of it, other things being equal, is a pretty good gauge of the condition of the heart muscle and, as will be seen, of the gravity of the disease. When the blood pressure in the kidneys is reduced the quantity of urine is lessened from 1,500 c.c. in the twenty-four hours to 800, 600, 400, or less. In the gravest cases no urine at all is passed. But if the in-

gestion of water continues and is no longer eliminated through the natural channels, it accumulates in the body and chiefly in the subcutaneous tissues, where it constitutes œdema, hydrops, anasarca. Any retardation of circulation in the kidney leads quickly to albuminuria, so that the diagnosis is often embarrassed in this way and may be cleared up at times only by the discovery of specific casts. Protracted stasis develops in the kidney the same hyperplasia of connective tissue as in the liver and the same characteristic "cyanotic induration."

The processes of *embolism* have been studied in detail under the subject of acute endocarditis, in which condition the accident is more liable to occur. At the same time it may be readily understood that thrombotic masses may at any time be detached from the valves or walls of the heart and swept off by the torrent of blood to lodge in some distant organ. These emboli in the course of chronic endocarditis are for the most part bland. They act therefore only in a mechanical way and produce in various organs the changes which are included under the term infarction. It was Virchow who first of all explained the process of embolization and illuminated in this way some of the darkest fields in connection with heart disease.

Embolism of the brain occurs most frequently in the arteries of the Sylvian fissure, so that the occurrence of the accident is recognized by aphasia with hemiplegia, which suddenly set in. The relation of chorea and the possibility of the production of certain cases by the deposits of multiple minute emboli in the brain have been already discussed. Certain it is that many cases of chorea occur in connection with endocarditis, but the modern tendency is to ascribe the irregular convulsive action rather to the action of toxins than to mechanical occlusions. Embolism of the retina shows itself in sudden blindness or in limitation of the field of vision.

Burgess reported a curious case of heart disease in a woman, pregnant six months, in whom all the symptoms disappeared on several occasions, leaving the patient for days in almost perfecthealth. The diagnosis was changed accordingly from time to time, but was finally determined on the nineteenth day of the disease and thirty-six hours before death as an embolism of the brain. Goyet reports a case of embolism of the central artery of the retina with suppuration of the entire eyeball in the course of an endocarditis ulcerosa, and a second case of recurrent endocarditis followed by cataract with chemosis and complete amaurosis.

Embolism of the lungs reveals itself by dyspnœa with anxiety, sometimes with convulsions, suffocation, and, when a great artery is blocked, by sudden death. Where the occlusion occurs only in smaller

vessels, the patient recovers from the shock to show the signs of infarction in pain in the side and especially in spitting of blood. Where a large amount of lung tissue is suddenly occluded, death may occur at once and may be attributed in these cases to shock. Many of the cases of sudden death in heart disease occur under the picture of pulmonary embolism. There is a sudden seizure of agonizing pain with the sense of suffocation; the heart beats tumultuously, the face becomes cyanosed, the eyes seem to start from the sockets; there is a moment of violent tetanic rigidity, followed by a few gasping respirations, which terminate life. Sometimes the end is precipitated by an attack of epileptiform convulsions.

In the bronchitis of brown induration of the lungs, the sputum is tinged with blood and looks like that of croupous pneumonia. Under the microscope it shows oval cells with vesicular-looking nuclei. The cells are brown or black with pigment which stains them uniformly or is enclosed in the form of masses and plaques. These cells are distinguished as the cells of heart failure. Whether they are to be regarded as cells of alveolar epithelium (Sommerbrodt, Hoffman) or leucocytes (Lenhartz) is still undetermined.

Embolism of the spleen is attended with the supervention of sudden pain in the side, with tenderness to pressure, with increase in the size of the spleen, and sometimes with the friction sounds of a circumscribed peritonitis in the region of the spleen. Fauvelle found embolism of the splenic artery in the case of a girl who suffered intense pains in the region of the spleen twelve days before death.

Embolism of the intestine is revealed by more or less marked colicky pain, which is usually severe, sometimes by dysentery with the discharge of blood.

Embolism of the kidney is attended also with pain in the region of the kidney, sometimes so severe as to simulate that of the passage of renal calculi, is marked at first usually with more or less arrest of function, and followed later by the passage of small quantities of urine, more or less distinctly tinged or mixed with blood.

Embolism of the extremities is attended with a sensation of numbness, formication, extreme coldness, and is sometimes, though very rarely, followed by gangrene. Trousseau speaks of the case of a young married lady whom he was seeing in consultation with a medical friend, and who during the consultation felt uncomfortable sensations in the region of the heart and was afterward suddenly seized with painful tingling in the fingers. The fingers had a bluish color and very soon presented all the appearance of a dry gangrenous affection; fortunately the gangrene was limited to one of the last phalanges, which the patient lost. Eighteen months later this lady

was suddenly stricken with complete paralysis of one side of the body, and subsequently sank with all the symptoms of softening of the brain.

Embolism of the skin reveals itself in multiple discolorations or eruptions of various character, most frequently petechial or hemorrhagic.

In all cases interference with the action of the various organs, especially with the organs concerned in the elimination of toxic products, leads to the accumulation in the body of these poisons, which show their presence in the blood in attacks of fever with all its concomitants, in defective cerebration, interference with the digestion, jaundice, oliguria, uræmia, etc. The beneficial action of the diuretics and of many of the heart stimulants which act in the same way is to be ascribed to the elimination of these poisons from the body.

Certain lesions may be more readily compensated than others, or the compensation is in certain cases more readily broken than in others. But in all cases the final result is the same. In the case for instance of mitral regurgitation the stasis in the lungs is overcome for a time, sometimes for a long time, by hypertrophy of the right ventricle. Finally, however, the hypertrophy must give way. Hereupon there must ensue the general signs of heart failure. In the case of aortic insufficiency the lesion is compensated for a long time by the extreme hypertrophy of the left ventricle. But this hypertrophy must also give way in the course of time. The left ventricle will then become dilated and incompetent. The same thing may be said of the other affections of the same valves or of other valves. The final scenes are the same. The effects differ, not in their character, only in the time of their occurrence.

We are now prepared to take up the study of the various valvular lesions in detail, and we may begin, as the most frequent of all the affections of the valves, with

Insufficiency of the Mitral Valves.

In a note appended to the description of Laennec, Andral (1827) (who died at the advanced age of seventy-nine of an old heart lesion) described with great precision the mechanism of mitral insufficiency, which term was then for the first time introduced into the medical vocabulary. When the valves, he said, no longer oppose the reflux of blood, they become insufficient, and this condition is known as an insufficiency of the valves.

Insufficiency at the left auriculo-ventricular orifice is that condition in which under contraction of the left ventricle the mitral valve fails

to close the orifice, so that some of the blood, all of which should have been propelled into the aorta, escapes into the left auricle whence it came. Endocarditis will have been the cause of the incompetency in the great majority of cases. The infection or inflammation thickens the valves or leads to deposits upon their surfaces, especially upon the ventricular surface, so that the necessary accurate coaptation of the two segments becomes impossible. In other cases the inflammation may be more strictly confined to the wall of the heart. The papillary muscles may have lost tone or the tendinous cords become thickened so as to bind down the valves and in this way prevent perfect closure of the orifice. In rare cases the valve may be split in consequence of strain or the rupture of an aneurysm, or a segment may be detached from the ring of insertion at the base of the valve. Barié collected thirty-five cases of localized ruptures of the valves, sixteen of which concerned the mitral valve. The condition is observed therefore, being the consequence of an infection, chiefly in youth and maturity, the period of prevalence of the originating maladies.

But sometimes insufficiency of the mitral valve is developed in old age. In these cases the mitral valve is, as a rule, infected secondarily, after the aortic valve, and in consequence of atheromatous change. Not at all infrequently some atheromatous degeneration occurs in the course of chronic endocarditis, so that patches of opaque and thickened tissue, sometimes chalky or gritty to the feel, occur in the structure of the valve or are displayed along its edge. In this way the valve loses its suppleness and elasticity, and even a slight thickening may be so disposed as to interfere with the free action of the valve.

In a third class of cases there may be no affection of the endocardium whatever, but the myocardium, especially in the vicinity of the auriculo-ventricular orifice, may have lost tone through inflammation so that the orifice is no longer properly adjusted to permit closure by even perfectly normal valves. As stated elsewhere, the auriculo-ventricular orifices, though circular or oval during the period of diastole, change their shape under the contraction of muscular fibres at the base of the heart to assume a more slit-like or button-hole appearance. Any impairment of the tone in these muscles, any paretic condition, for instance, under acute inflammation of the myocardium, or any cicatricial contraction in the course of chronic myocarditis, may thus interfere with the natural alteration in the shape of the orifice necessary to secure perfect occlusion by the valves. In any case an hypertrophy which may have compensated a valve lesion or have remedied a defective tonicity gives way in the course of time, so that the heart muscle becomes weak and the heart cavities undergo dilatation under the continued pressure of the blood. This dilatation may

become so great that the valves no longer suffice to close the orifice. Such an incompetence is distinguished as a relative insufficiency. This relative insufficiency is more common in the right heart, but is observed quite frequently in the left heart in the later course of the hypertrophies which arise in consequence of increase of blood pressure, arteriosclerosis, Bright's disease, etc. Pellegrini described a typical case of relative insufficiency at the mitral orifice caused by fatty degeneration of the papillary muscles. The patient was a man, aged sixty, in whom a systolic murmur could be heard over the hypertrophied left ventricle. The autopsy revealed perfect valves with fatty papillary muscles and arteriosclerosis as the cause of the hypertrophy.

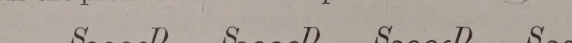
Finally insufficiency may be caused by a neoplasm. Thus Curtis found in the case of a woman who died suddenly a myxoma on the auricular face of the posterior leaflet of the mitral valve. A similar lesion was found by Debove in another case, on the tricuspid valve. But from whatever cause occurring—from endocarditis, including under this head thickenings and deposits upon the surface of the valves, stretching or rupture of the tendinous cords, paresis or paralysis of the papillary muscles; from atheromatous degeneration, rupture of aneurysms, or from muscular strain; from relative insufficiency in the course of chronic myocarditis and dilatation—the general effect is always the same; some of the blood regurgitates during the systole into the left auricle, so that only part of the contents of the left ventricle is discharged into the aorta.

Symptoms.

The reflux of blood from the left ventricle into the left auricle develops a murmur which is heard with the greatest intensity at the apex and which is necessarily isochronous with the systole, that is, with the first sound of the heart. This murmur is usually soft or blowing in character and much resembles the whispered "who," save that it is much longer drawn, as a rule, being often protracted quite up to the period of the second sound. Hope distinguished the bruit of mitral insufficiency as rough or soft, according to the nature of the lesion, and usually very low in pitch. It may, however, have any variety of timbre. It may show even a musical note and sometimes a peculiar screaming sound, the so-called *bruit de piaulement*. Lépine found on autopsy, in a case in which this bruit had been very distinct, a perforating aneurysm with anfractuous walls. Sometimes the murmur sounds like the escape of a jet of steam. In other cases it is as rough as the sound of a saw or rasp, or it may vibrate like a rattle or with the thrill of the jews-harp. In one such case Potain and

Rendu found at the autopsy a tendinous cord detached from the rest and stretched across the ventricle wall in such a way as to traverse the cavity and form an obstacle to the column of blood (Petit).

The bruit, being caused by the regurgitation of blood under the contraction of the left ventricle, should correspond exactly in time to the length of the systole and should terminate when the contraction ceases. Usually the bruit is heard most intense toward the close of the systole at the moment of final forcible contraction; sometimes the murmur runs so close up to, that it seems to be prolonged into the second sound produced by the closure of the valves at the base. Paul calls this bruit the *souffle paradoxal* because it appears to belong to the diastole while it really belongs to the systole. Fraentzel calls this murmur the prediastolic. He represents it with this simple drawing:



Fraentzel declares that he has heard this murmur at least twenty times and has always been justified in concluding that it depends upon insufficiency of the mitral valves. Anything like a presystolic murmur belongs of course to a coincident or complicating stenosis.

Sometimes the bruit, which is distinctly synchronous with the first sound, is heard in great, if not in equal, intensity at the base of the heart to the left of the sternum in the region of the sounds of the pulmonary valve. The recognition of the bruit in this region was for a long time a puzzle to clinicians and its localization met with various explanations. It was at one time ascribed to greater tension in the walls of the pulmonary artery and to closer approximation of the artery to the walls of the chest from displacement of the left lung, which left it more uncovered. But when it was observed that the bruit is not heard in cases of mitral stenosis in which the tension of the pulmonary artery is greatest, this view had to be surrendered.

Naunyn offered the most satisfactory explanation, which not only covers the cause of the anomaly, but also the fact that it is observed in some cases and not in others. Naunyn shows in the first place that the sound is heard best not exactly in the region of the pulmonary valve sounds, that is, not directly at the line of the costo-sternal junction, but about two inches to the left of it. This region corresponds to the point of the appendix of the left auricle, which here insinuates itself around the pulmonary artery and comes to lie in front of it. Now, as the blood is propelled by the left ventricle through the incompetent mitral valves into the left auricle, it is easy to understand how the sound of the murmur may be propagated to the left auricle and be heard especially over the appendix, which lies immediately under the ear in this region. The absence of the sound in many

cases is due to the fact that the auricular appendix is not long enough to surround the pulmonary artery and lie in front of it. On the contrary, it stops short and lies too deep to transmit to the ear of the auscultator the propagated sound. Curschmann comments upon the frequent occurrence of this systolic murmur at the base, at the point of auscultation of the pulmonary artery, instead of at the apex. Curschmann considers it no accident but a really valuable diagnostic sign, which in a doubtful case speaks for an organic and against an accidental murmur. The condition occurs in those cases of recent mitral insufficiency in which the dilatation of the right ventricle exceeds that of the left to such degree that the left is more than normally pressed away from the wall of the chest and lies against it only with the enlarged auricle. The murmur is therefore audible exclusively or chiefly at the point of projection of the auricle, while it may be scarcely perceived at all at the apex.

It is worthy of remark that the murmur over the pulmonary artery may be better heard under certain changes of posture, as for instance in lying down.

As the blood is regurgitated into the left auricle, this cavity becomes dilated. Some attempt is made to overcome the pressure by hypertrophy, but the capacity of the auricle in this direction is slight. The hypertrophy is itself soon overcome, the left auricle dilates to some extent, and not infrequently this dilatation may be recognized by increase of dulness higher in the thorax, sometimes up to the level of the second rib, under and on the left side of the sternum.

The rise of blood pressure in the left auricle necessitates a like rise of pressure in the pulmonary veins and, following the circulation backwards, in the pulmonary artery, and this elevation of pressure in the pulmonary artery gives rise to one of the most valuable and distinctive signs of mitral insufficiency in a direct intensification of the sound of closure of the pulmonary valves. For as the blood is propelled by the right ventricle into the pulmonary artery, which is already distended, the resilience of the artery forces the closure of the pulmonary valves under high pressure. In fact, the pressure is so great and the closure of the valves so sudden as markedly to intensify the second sound of the heart. This intensification of the second sound, which is technically known as an accentuation, is a direct consequence of the distention of the pulmonary artery. The great value of this sound becomes apparent at once and every observer applies the stethoscope at the point nearest to the pulmonary valve, that is, at the junction of the second rib with the sternum or in the second interspace to the left of the sternum, to ascertain the pitch of

this tone and to compare it with the sound of the closure of the aortic valves at a similar point on the right side of the sternum. This accentuation or intensification of the pulmonary valve sound indicates of course only a higher pressure in the pulmonary artery. The rise of pressure may be due to obstruction in the lungs or to sclerotic changes in the walls of the vessel. These are conditions, however, which are not usually noticed in the course of heart disease or they may be eliminated by distinctive signs, so that the accentuation of the pulmonary valve sound which was first signalized by Skoda, who at once appreciated its importance, becomes a cardinal sign in the recognition of lesions of the mitral valve. The accentuation may be heard as long as the increase of pressure is sustained in the pulmonary artery, that is, as long as the compensatory hypertrophy of the right ventricle lasts. It is only when this hypertrophy begins to flag that the accentuation of the pulmonary valve sound may be no longer perceived, but with the disappearance of it a sign of inestimable value in the diagnosis of mitral insufficiency is lost.

The propulsion of the blood wave against the higher pressure in the pulmonary artery soon develops hypertrophy in the wall of the right ventricle, which is appreciated as an increase of dulness beyond the right border of the sternum. This hypertrophy becomes so pronounced as entirely to overcome the additional pressure in the pulmonary artery and in the pulmonary veins, so that the power of the right ventricle is invoked in aid of the weak left auricle. When the ventricle is able to counteract the effect of the regurgitation at the auriculo-ventricular orifice, the hypertrophy is said to be compensatory. A sufficient quantity of blood is then propelled as if by a double force pump, one quite weak it is true, into the left ventricle, so that notwithstanding the defect, the aorta is kept amply supplied with blood. Such an hypertrophy occurs, as a rule, and remains often for a long time—so long, indeed, that years may lapse without the discovery of the condition. In fact, the lesion is often made manifest only in the presence of some extraordinary effort, some intercurrent disease, or some depressing emotion which weakens the myocardium and breaks up the perfect compensation.

The increase in volume of the right ventricle necessarily changes the shape and position of the heart in the thorax. The hypertrophy of the right ventricle brings the heart to lie more upon its side, so that a greater surface rests upon the diaphragm. The powerful pulsation of the right ventricle may be perceived at the ensiform cartilage. The increase in volume necessarily also dislocates the apex of the heart to the left. Sometimes the hypertrophy of the right ventricle is so great as to overshadow the left. The right ventricle

in these cases forms the apex, which may be felt as far over as the axillary line. This changed relation of the right and left ventricle and the dilatation of the right heart and retreat of the left are exemplified by Curschmann in the accompanying cut, which represents a

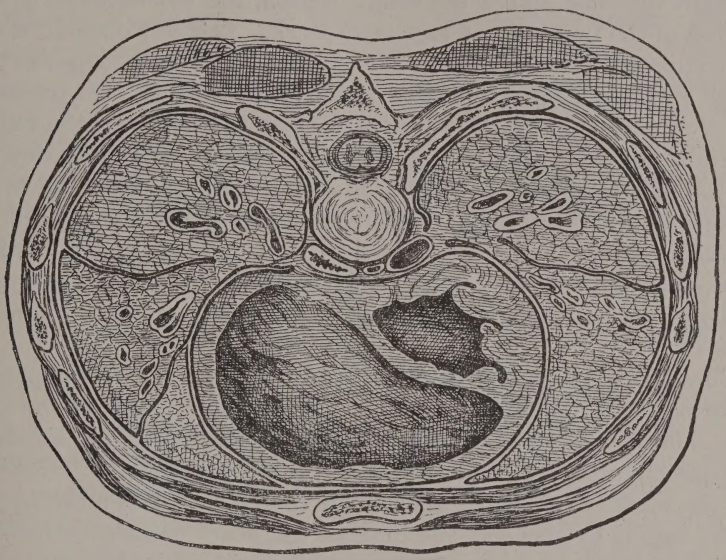


FIG. 6.

cross section through the frozen body of a boy who died suddenly during the course of a chorea with recent mitral insufficiency.

The conduct of the left ventricle in these cases is a matter of dispute. Sometimes it is larger than natural, sometimes less. Strümpell maintains that the work of propelling the blood through the incompetent valves brings about a distention of the left ventricle, that is, the work of the left ventricle is increased by the fact that the blood finds issue through two orifices. Eichhorst and Fleischer coincide with this view. On the other hand, Friedreich never noticed any hypertrophy of the left ventricle, and Fraentzel maintains that any enlargement that may exist affects only the left auricle and not the ventricle.

Any enlargement of the left ventricle in insufficiency of the mitral valve has been considered by other authors as inconstant, or when present as the consequence of other conditions, affection of the kidneys, arteriosclerosis, etc. The supersaturation of the blood with carbonic-acid gas has also been invoked (Traube, Cohnheim) to account for the increased tension in the arterial system. In the presence of a leak, the left ventricle receives less blood than normal,

or if it receive the normal amount of blood must expend less effort in expelling the blood from its cavity, because the blood may escape in two directions. Ordinarily, therefore, the left ventricle having rather less to do, undergoes no hypertrophy. When the hypertrophy of the right ventricle makes the compensation, the left ventricle is still relieved of the necessity of any extra work. In a few cases in which this compensation is excessive, the left ventricle may undergo hypertrophy in slight degree to overcome the strain of additional pressure. But when the compensation begins to flag, that is, when the wall of the right ventricle begins to suffer degeneration and dilatation, so that blood accumulates in increasing quantity in the lungs and in the left auricle, the increase of pressure makes itself felt also in the left ventricle, which now in turn suffers degeneration and dilatation. Finny reported a case of mitral regurgitation with extreme hypertrophy of the left ventricle and embolism of a branch of the dorsalis pedis artery.

These various phases may be felt in the practice of palpation. Under perfect compensation the impact and the position of the apex stroke may be perfectly normal. Under anything like an excessive compensation the heart may be felt to throb with greater force and the apex strikes with vigor the wall of the chest. When the hypertrophy begins to give way the force of the heart becomes enfeebled, the impact may be no longer felt, and the apex stroke becomes weak or imperceptible. Under increasing dilatation the walls of the right ventricle may be stretched so that the tricuspid valve becomes relatively insufficient. The pulsations of the heart may be then seen in the veins in the neck and may be felt in the substance of the liver.

During the stage of compensation the pulse shows no anomalies. The hypertrophied right ventricle forces the blood back through the

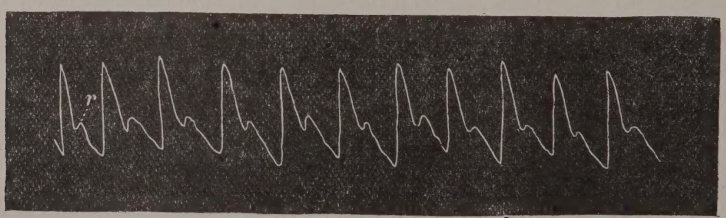


FIG. 7.—Pulse Curve of Mitral Regurgitation with Perfect Compensation. *r*, Recoil.

pulmonary artery, the capillaries of the lungs and the pulmonary veins into the left auricle and left ventricle, so that, notwithstanding the regurgitation, the left ventricle may propel into the aorta a sufficient amount of blood to fill the aorta and sustain the natural pres-

sure throughout the arterial system. It is only when the right ventricle begins to flag that the pulse shows alterations in its force and rhythm. The pulse now becomes irregular and arhythmic or shows distinct intermissions. In advanced cases some of the contractions of the heart may fail to reach the radial artery.

The finer variations are traced by the sphygmograph. But the tracing in these cases depends entirely upon the condition of the heart muscle. The tracings of mitral insufficiency usually show a short line of ascent with distinct dicrotism on account of the laxity of the arterial wall under incomplete distention. The irregularities sometimes assume a definite form, so that the arhythmia becomes an allorrhythmia, which has been studied in detail under the subject of arhythymia.

Diagnosis.

The diagnosis of mitral insufficiency is usually determined without much difficulty. It rests upon the following facts:

(1) A bruit synchronous with the first sound and heard in greatest intensity at the apex of the heart. In certain cases, as stated, this bruit may be heard in great or nearly equal intensity at the base of the heart to the left of the sternum, for it is propagated into the left auricle and conducted through the auricular appendix as it surrounds the pulmonary artery and lies close under the wall of the thorax.

(2) Hypertrophy of the right ventricle as indicated by increase of dulness to the right of the sternum and dislocation of the apex to the left. There is also some slight hypertrophy of the left auricle which may make itself manifest by increase of dulness in the neighborhood of the second rib on the left of the sternum.

(3) Accentuation of the pulmonary valve sound, which is due, as stated, to the hypertrophy of the right ventricle, and which persists so long as the hypertrophy lasts. This accentuation is heard directly over the valves in the second interspace on the left, or at the junction of the second rib with the sternum.

These are the three cardinal signs in the diagnosis of insufficiency of the mitral valve.

As elsewhere stated, a murmur at the apex in the course of the first sound of the heart may meet with various interpretations. In the first place it may be entirely independent of any lesion of the valves whatever, or of any organic affection of the muscle of the heart. It may be due simply to anæmia and to the defective nutrition which this condition implies, that is, to the irregular vibrations of the blood current and the walls of the heart. A bruit with the first sound may also depend upon stenosis at the aortic valve, though here

the bruit is most marked at the base. Finally, the pericardial friction is more apt to coincide with the first sound of the heart. The mere discovery of a murmur at the apex synchronous with the systole by no means indicates of necessity an affection of the mitral valves.

Anæmic (accidental) bruits are distinguished by the genetic relationship to anæmic states (chlorosis, leukæmia, etc.), independent of the infections which produce endocarditis, or, as not much stress can be laid upon this point because anæmia occurs regularly in the course of valve lesions, by the absence of signs of enlargement of the heart and accentuation of the pulmonary valve sounds.

It is often impossible to determine whether insufficiency be absolute or relative. A relative insufficiency at the mitral valve occurs in the course of the so-called idiopathic hypertrophy of the heart, when the hypertrophy begins to give way and dilatation ensues. The genetic relationship of the condition may make the diagnosis. Thus idiopathic hypertrophy occurs more especially as the result of age, arteriosclerosis, Bright's disease, gout, diabetes, hard work, etc.; whereas the absolute insufficiency depends in the vast majority of cases upon endocarditis. The physical signs may be the same in either case.

The pericardial friction sound is heard usually in greatest intensity at the base, does not correspond so exactly with the phases of the action of the heart, is altered by changes in posture, by pressure, exercise, etc., and comes and goes, while the endocardial bruit stays.

Prognosis.

The prognosis of mitral regurgitation is the most favorable of all the lesions of the valves. These are the cases in which the nature of the disease may escape recognition for years, and individuals thus affected may live for many years, even decades of years, without any signs which point directly to disease of the heart. This is especially the case in childhood, when the hypertrophy of the right ventricle completely compensates the defect in the valve. The prognosis depends also upon the rapidity of development of the lesion. As a rule, the origin is insidious and the development is slow, so that the heart may adjust itself to the lesion and tolerance is begotten to the disturbance in the circulation. But when the insufficiency develops rapidly, as in the case of rupture of the valve, the signs are very severe and the prognosis becomes correspondingly grave.

In six of the thirteen cases recorded by Barié, rupture of the mitral valve was announced by intense pain in the chest with extreme dyspnœa, amounting almost to suffocation, the face became livid, the

body was covered with a clammy sweat, the extremities were cold, the heart fell into delirium. In a case of this character Stokes discovered upon autopsy rupture of the tendons of the mitral valve.

The duration of life after sudden rupture varies between eight days and twenty months. It is seen that death is never sudden, though Allix reported a case in which life lasted but one hour, Hanot another case in which life lasted several hours only. The prognosis becomes bad in proportion as the regurgitation is complicated by stenosis, but depends especially upon the degree of affection of the myocardium.

The prognosis depends in great extent upon the care which the patient is able to take of himself. Many cases survive to succumb to old age, but most cases are cut short by intercurrent maladies. Any acute disease, especially any affection of the lungs, aggravates the prognosis at once. The subject is considered again under the general subject of "Prognosis of Heart Disease," where the "Treatment" is discussed at length.

Mitral Stenosis.

The history of stenosis of the mitral valve begins with Corvisart (1818), who noticed that of the two auriculo-ventricular orifices, the left is most frequently the seat of contraction by induration and ossification. Corvisart recognized by palpation active palpitation and irregular action with arhythmia of the pulse. At an autopsy he found the left auricle distinctly dilated. The auriculo-ventricular orifice was extraordinarily contracted and formed a kind of osseous slit through which only a small silver piece could be forced. Laennec laid stress upon the peculiar vibration *bruissement*, which was first noticed by Corvisart when the hand was applied to the precordial region, and distinguished it as the *frémissement cataire*, the name by which it is still known (Petit).

Obstruction of the left auriculo-ventricular orifice is a more correct expression than mitral stenosis, because it has more general application. The lesion which contracts or occludes the orifice does not necessarily affect the valves at all. Nevertheless in the majority of cases the obstruction is caused by deposits upon or by thickenings or adhesions of the mitral valves. In rarer cases a protruding aneurysm of the wall of the ventricle or of the valve itself, or in still rarer cases a tumor, that is, a neoplasm, may be so situated as to offer obstacle to the current of blood. B. Curtis and T. Curtis have each reported a case of tumor of the mitral valve.

In rarer cases extensive exuberations from the aortic valve may

reach down into the left auriculo-ventricular orifice and bring about a stenosis. Fraentzel and Barnhardt both observed cases of this kind.

The obstacle in obstruction at the left auriculo-ventricular orifice interferes with or retards the escape of blood from the left auricle into the left ventricle. Such a lesion rarely exists alone. As already stated, insufficiency of the mitral valve is usually complicated with some degree of stenosis, though as a rule the stenosis is so slight as to offer no real difficulty in the complete emptying of the auricle. A mitral insufficiency may exist alone. A mitral stenosis is almost never found alone; it is nearly always associated with mitral insufficiency. The lesion which offers obstacle to the escape of blood from the auricle is so disposed as also to prevent perfect closure of the valves and permit regurgitation. As a rule, therefore, the condition which is usually encountered is obstruction with regurgitation. The obstruction assumes prominence when it distinctly interferes with the escape of blood from the left auricle, and when in these cases the regurgitation is so slight that it may be practically overlooked, the lesion is distinguished as an obstruction at the left auriculo-ventricular orifice, or for the purpose of practical description as a mitral stenosis.

The lesion which produces the obstruction is usually the same as that which prevents the closure of the valves, namely, a chronic endocarditis. Tough, fibrinous, thrombotic masses, deposited upon the surface of the valve and involving the tissue of the valve itself, protrude so as to obstruct the circulation of blood. More frequently the valves are thickened into a fibrinous mass or are rolled up along the free edges so as to act in the same way. In still other cases atheromatous degeneration has set in and calcareous thickenings occur in the surface of the valve and oppose an obstacle to the circulation of the blood. Chaubasse and Labranche reported a case of complete calcification of the mitral valve, also of the left auricle, with extraordinary dilatation of the left ventricle, and Gaucher reported a mitral lesion with complete adhesion of the valves, which were united to form a funnel. These changes may take place in the course of chronic endocarditis or may result from an extension of the atheromatous change from the aortic valves. The processes of endocarditis occur more especially in younger life or in the course of some infection, especially rheumatism. The atheromatous changes occur more frequently in more advanced life in connection with arteriosclerosis. Sometimes the deposits or thickenings are ranged about the ring of insertion of the valves. Féréol reported a case of calcification of the fibrous zone of the mitral valve. Sometimes the deposits

are seen in the body of the valve itself, but more frequently they are found along the free edges. Sometimes the shrinkage of the tendinous cords is so great as to bring the thickened valves to rest immediately upon the papillary muscles.

The size of the auriculo-ventricular orifice may be roughly estimated by the fingers, as ordinarily both the index and middle fingers may be passed through the opening. But it must be remembered that post-mortem rigidity may much contract the orifice and render this estimate invalid. Disease processes may narrow the ring so that only one finger or a goose quill may be pushed through.

The normal width of the orifice is so great that a moderate contraction offers no marked obstacle. Even a diminution to one-half of the normal width imposes but little extra work upon the left auricle and the right ventricle. It is only when the contraction exceeds this limit that hypertrophy must take place to compensate the lesion, and compensation becomes impossible only when the orifice is reduced to one-tenth of its normal width.

As in the majority of cases mitral stenosis is produced by endocarditis, especially that form which occurs in consequence of rheumatism, the condition is most frequently encountered in early life. In fact mitral stenosis has been found at birth. Deschamps reported a case of congenital mitral stenosis. Sansom collected 40 cases in infancy, 19 of which were verified at the autopsy. Edwards reported 12 cases of mitral stenosis in children, one of them a boy aged five and one-half years. Landouzy declared that the condition was rather more common in the female sex, 76 to 100.

Symptoms.

The general symptomatology of mitral stenosis is in the main the same as that of mitral insufficiency, and all the more is this the fact because the two conditions are, as a rule, conjoined; at least mitral stenosis is found nearly always in association with mitral insufficiency. Mitral stenosis is in a general way a graver lesion than mitral insufficiency, and the symptom which develop in consequence of any valve disease set in sooner and in more severe form, so that while many cases may be announced with the signs of a general failure, or symptoms of affection of individual organs may show themselves in greater intensity, attention is sooner directed to the heart itself in mitral stenosis than in mitral insufficiency.

Evidence of stasis in the lungs shows itself first upon exercise or after some emotional disturbance. The patient finds his breath short in climbing stairs. He takes cold on the slightest exposure and the cold is exceedingly obstinate. There is cough with the expectoration

at first of frothy mucus, later of mucus tinged with blood. There may be later hæmoptysis from more extensive exudation or as the result of infarction. The signs of defective blood supply to the brain set in sooner. There is difficulty in concentration of the ideas. There is unwonted apathy with emotional disturbance; the patient becomes irritable, irascible, and morose. The face is pallid, the mucous membranes more or less anæmic at first. Later the veins are ectatic, the face becomes dusky, in the later stage of the disease, cyanotic. The veins of the neck are distended and show undulatory motion and pulsation. The liver is swollen, the mucous membrane of the bile ducts is thickened, causing icterus, and the quantity of urine is distinctly diminished. Dropsy accumulates sooner about the feet and mounts up more rapidly to the trunk. In short, all the signs of venous stasis show themselves sooner and are more severe.

As obstacle is offered to the escape of blood from the left auricle, the latter becomes dilated and makes what effort it may by increase in size to overcome the difficulty. As, however, the muscular tissue in its walls is but scantily developed, the auricle soon gives way, distending easily—apparently with the ease of a soap bubble—until it reaches its utmost limits. In consequence of this distention the blood is dammed back in the lungs so that the pulmonary vessels are kept in a state of extreme ectasia, to such a degree, indeed, as to bulge into and largely encroach upon the space for the air; hence the signs of early interference of the aëration of the blood in mitral stenosis, duskiness, venous distention, and cyanosis. Hence also the defective nutrition of the lungs and the predominant tendency to catarrhal affections and to hemorrhage.

When the lung capillaries are overfilled the pulmonary artery is distended, and the blood pressure is raised to such a degree as to shut the valves suddenly and with distinct accentuation. The right ventricle now undergoes hypertrophy and this change sets in sooner and advances, that is, yields more rapidly than in the case of mitral insufficiency. All these changes are precipitated and intensified in this condition, for the simple reason that in mitral stenosis the blood accumulates and is retained in the left auricle in greater quantity than it is in mitral insufficiency. Dilatation of the auricle sets in sooner, ectasia of the lungs is more extreme, the pressure in the pulmonary arteries is higher, and the hypertrophy of the right ventricle is more rapid and pronounced.

These various changes make themselves manifest by physical signs, so that mitral stenosis may be as a rule readily recognized.

Physical Signs.—Upon inspection the chest may show nothing abnormal, but in many cases the hypertrophy of the right ventricle

makes itself manifest by increased impact, especially by more distinct pulsation of the epigastrium, and by more pronounced dislocation of the apex to the left. Thus the apex may be seen to strike the wall of the chest half-way between the mammillary and axillary lines. In youth the wall of the chest may be protruded.

But when the hypertrophy begins to give way the pulsations of the heart become more feeble and the impact of the apex, as well as the body of the heart, may not be seen at all. Thus, during the stage of perfect compensation nothing abnormal may be observed at all. In the last stage of the disease, when the compensation is entirely broken, the heart is too feeble to make its pulsations manifest to the eye. The one sign then visible to inspection is the more pronounced palpitation which is so commonly observed at the ensiform cartilage from the preponderating dilatation of the right ventricle.

These movements or lack of movements of the heart may be more distinctly appreciated by palpation. Under good compensation as a rule palpation may perceive nothing abnormal. At other times the tumultuous action or dislocation may be recognized by palpation when invisible to the eye. Not infrequently a distinct vibratory thrill may be perceived by the hand applied over the body of the heart or over the apex. This thrill is the *frémissement cataire* of Laennec, which was first observed by his distinguished preceptor, Corvisart. It was in mitral stenosis that Corvisart first noticed the vibratory thrills which have since been so frequently connected with obstructive lesions. The feeling as described is exactly like that of the purr of a cat or like the vibration communicated to the hand by a blow upon a spiral spring of a sofa or cushioned chair.

Percussion shows dulness extending upward often as far as the second rib over the dilated auricle, to the right beyond the sternum, and to the left beyond the mammillary line. As compensation lasts but a comparatively short time, the increase in dulness is usually due to dilatation.

Still more definite information is furnished by auscultation. When the ear is applied to the surface of the chest in the neighborhood of the apex a distinct murmur may be heard in connection rather with the second than with the first sound of the heart, the murmur caused by the passage of the blood through the constricted orifice. The actual time of its occurrence will depend upon the character and extent of the lesion. When the orifice is not too small, the first part of the contents of the auricle falls by gravity through the open mitral valves. The real obstacle is offered to the escape of the last part of its contents under what contraction the auricle may make. The friction sound is greatest as the auricle attempts to force

the blood through the contracted orifice, just at the end of the diastole and just before the systole. Such a murmur is said, therefore, to be presystolic. It does not occur exactly at the moment of contraction of the ventricle nor at the moment of beginning dilatation, but corresponds to the end of the period of dilatation which immediately precedes the contraction of the ventricle. This difference in time may be best appreciated by palpation of the pulse during auscultation. The pulse corresponds to the period of systole or is slightly, almost inappreciably, later than the systole. The murmur occurs just before the systole, is really presystolic, so that the three periods which belong respectively to the murmur, the systole, and the pulse make themselves manifest successively to the ear and to the touch.

The murmur is heard in greatest intensity at the apex. Sometimes this point is shifted a little to the left because the left ventricle in which the murmur is formed is displaced slightly backward and to the left by the enormously enlarged right ventricle.

The murmur results from the forcing of blood through the contracted orifice. It must be therefore presystolic. The systole stops it. Sometimes the systole furnishes a murmur of its own in consequence of a coexisting insufficiency, but in the absence of this insufficiency the first sound is pure and, in fact, is stronger than natural.

Liebermeister attributes the strengthening of the first sound at the apex to the conduct of the segments of the mitral valve. In the normal relation the mitral valves at the beginning of the systole are apposed so that the valve stands in the position of closure. The effect of the systole is simply to stretch the segments and to bring them into closer apposition. In stenosis of the orifice the influx of blood from the auricle into the ventricle keeps the valves open up to the moment of the systole; thereupon at this moment they are suddenly closed. The segments must make, therefore, unusually wide excursions. Liebermeister declares that he has repeatedly noticed such increase in the first sound that it could be heard at some distance.

Potain, too, remarks upon the fact that the first sound of the heart is particularly hard, but he attributes it to the stiffness of the mitral valves. Potain speaks also of a *claquement* attending the opening of the mitral valve, a sound which arises in the beginning of the diastole under the distention by the current of blood of the stiff sections of the mitral valve, which are also partly adherent to each other along their borders. This sound may actually simulate a split of the second sound of the heart or may exist beside the split second sound.

Finally, Fenwick and Overend find the cause of the strengthening

of the first sound not in the mitral valves at all, but in the more powerful closure of the tricuspid valve under the hypertrophy of the right ventricle because, they say, the sound is stronger not at the apex, but to the right of it; moreover, because it is propagated to the right; and finally and especially, because it disappears so soon as the tricuspid valves become incompetent.

The right ventricle undergoes distinctive hypertrophy. But what is the conduct of the left ventricle? Here again observers differ. It is said on the one hand the left ventricle receives but little blood and easily discharges its contents into the aorta. Why then should it increase in size? Dunbar defends this view when the stenosis is pure. The fact is, however, that the stenosis is almost never pure; it is almost always associated with regurgitation, so that the ventricle shows the changes which belong to mitral insufficiency. In fact, Baumbach declares that he found atrophy of the left ventricle only seven times in ninety-seven cases of mitral stenosis, and in only one of these cases was the stenosis pure; it was always complicated with other lesions. So far from undergoing hypertrophy the left ventricle, then, as a rule, suffers atrophy. It accommodates itself to the diminished demands. Under these conditions degeneration soon sets in and becomes more extreme on account of insufficient nutrition of the heart through the coronary arteries. But Lenhartz denies the universal occurrence of atrophy. Lenhartz found at autopsy in a case of anatomically pure mitral stenosis a moderate dilatation of the left ventricle, while its wall was of at least normal thickness. The autopsy reports of the Pathological Institute at Leipsic and Halle concerning 97 cases of mitral stenosis showed in only 7 the left ventricle small and atrophic, while in 30 cases the ventricle was hypertrophied or dilated. In 10 cases (11 with his own) there was no complication with other valves, and mitral insufficiency could be absolutely excluded. Of these cases only 1 showed a thickness of 9 mm., while in the rest the thickness of the wall of the heart varied between 1 and 1.8 cm. and the cavity of the heart was only rarely contracted. It was more frequently normal or dilated. Lenhartz concludes that the compensation by mitral stenosis is more favorable than is usually believed, and expresses the view that perfect filling of the left ventricle is guaranteed by a suction action of the heart, which secures an hypertrophy of the left ventricle.

Sondheimer, of Heidelberg, saw in the course of twenty years 4 cases of pure stenosis, 6 of stenosis with insufficiency, and 2 of stenosis with aortic lesions. The condition of the left ventricle varied in the different cases. Contraction of high degree is usually attended with atrophy and reduction in size of the chamber of the left ventricle with

subsequent dilatation. When the stenosis supervenes upon insufficiency, the hypertrophy of the insufficiency remains even in the presence of quite considerable stenosis. The increased "suction force" of the ventricle protects it from wasting. This idea of a suction force sounds at the first hearing like a perversion of the physiological process of dilatation.

Reference has been made already to the intensification of the pulmonary valve sound under the extreme hypertrophy of the right ventricle and distention of the pulmonary artery which occur in consequence of stasis in the lungs. The fact is that this accentuation is often so pronounced as to split the second sound at the base.

In the state of health the semilunar valves of the aorta and pulmonary artery close at the same time or so nearly at the same time that no difference may be appreciated by the ear. Any disturbance in the blood pressure in the pulmonary artery or aorta disturbs this relation. If the pressure be increased in one and diminished in the other, as is the case in lesions of the mitral valve, the disturbance is all the more pronounced. Attenuation of the aortic sound may be, therefore, a sign as valuable as accentuation of the pulmonary sound. At any rate, the weakening of the aortic sound makes more manifest the intensification of the pulmonic sound. In the case of mitral stenosis thoroughly compensated by hypertrophy of the right ventricle, the pressure in the pulmonary artery is extremely great. The process of opening the semilunar valves against pressure is therefore slower, and the closure of the valves under the rebound of the blood is, though accentuated, distinctly retarded, so that the loud pulmonary sound is heard later than the less distinct aortic sound.

The sounds of the heart are then represented by one long sound, the systole, and two short sounds, that of the closure of the aortic and pulmonary valves respectively. Peter calls this rhythm the bruit of the dactyle, and Petit shows that the rhythm is exactly the inverse of the bruit of the galop, which is represented by the anapest, consisting of two short sounds and one long sound. Potain insists upon it that the asynchronism of the valves is not always in the order of aortic precedence. The excess of tension in the pulmonary artery entrains, he says, the precession of the sigmoid closure in the right heart. But Geigel attributes it to the fact that the two large vessels are not simultaneously filled, as the excessively distended and less elastic pulmonary artery contracts a moment later than the poorly filled aorta. However this may be, the fact of the separation or reduplication of the sound remains to constitute one of the most valuable signs of mitral stenosis. The splitting of the sound at the base, that is, the double second sound, is heard most distinctly over the pul-

monary arteries, but may often be appreciated everywhere over the heart, and both the accentuation and reduplication not only may be audible, but may be actually palpable to the touch. The pulmonary sound is heard more distinctly, as has been stated, because the aortic sound is so faint.

But when the splitting of the diastolic sound is heard most distinctly at the apex of the heart another explanation becomes necessary, because the question here must concern the mitral valves. The simplest explanation of this splitting of the second sound, which is so important to the diagnosis of mitral stenosis, is now the following:

As the pressure sinks in the left ventricle after the systole, while it is raised in the auricle on account of stasis, and is still further raised on account of contraction of the auricle, there occurs at the period of diastole a stretching and tension of the mitral valve toward the ventricle. And this stretching makes a sound if the valve has not become too stiff to yield a sound; premising always that this sound is not concealed by a murmur. The absence of this split of the second sound in mitral insufficiency is explained by the difference in pressure in the ventricle and auricle, as in insufficiency the valves may be applied without sound to the wall of the ventricle and the blood flows unhindered through the easily unfolded valve (Neukirch, Pel).

Returning to the pulmonary valves, it is observed that the long-continued high pressure finally leads to dilatation of the pulmonary artery with relative insufficiency of the pulmonary valves. It was Pawinski who observed, besides the well-known symptoms of mitral stenosis, in certain cases a long-drawn blowing murmur which some times occupied the whole systole and sometimes followed the diastole. This murmur is most intense at the left border of the sternum in the third or fourth intercostal space; it is weaker in the second intercostal space and in the neighborhood of the parasternal line. It does not extend beyond the right border of the sternum, and is entirely absent over the aorta. The explanation is this: The blood pressure in the pulmonary artery reaches an extreme degree, in consequence of the obstacle in the pulmonary circulation which is caused by extreme mitral stenosis or by changes in the bronchi and lungs. When the right ventricle is at the same time in a state of hypertrophy and is sufficiently powerful, and the blood wave is discharged during the systole from the right ventricle into the pulmonary artery, the reflux is so violent as to dilate the wall of the artery, including the orifice. The semilunar valves now fail to close accurately and the regurgitation of the blood into the ventricle produces a diastolic murmur. This murmur furnishes a favorable prognosis, for the reason that it occurs only when the right ventricle is powerful.

But the character of the second sound, and the same may be said of the first sound, will depend altogether upon the condition of the heart muscle. The murmur is heard at the apex presystolic and the sound of the pulmonary valve is intensified at the base, or the sounds of the closure of the pulmonary and aortic valves are actually separated or split in two only in cases where the heart muscle is in good condition, that is, where there is compensatory hypertrophy. So soon as the hypertrophy begins to flag the murmur at the apex becomes less pronounced and the split second sound or the intensified pulmonary valve sound may no longer be heard. In fact, in the course of time the heart falls into delirium cordis with multiple irregularity and insufficient contraction so that at this time a definite lesion cannot be determined.

The apex murmur is not always so distinctly presystolic; sometimes it is more distinctly diastolic. It may, in fact, show itself in two forms: as a diastolic murmur at the beginning of the diastole, and as a presystolic murmur at the end of the diastole. Sometimes these two forms are combined, so that we may hear a distinct murmur at the beginning of the diastole, then again, after a short pause, at the end of the diastole immediately preceding the systole and cut off by it. Sometimes it seems to take the place of the first sound; sometimes it is prolonged beyond it. Here, however, are complications with mitral insufficiency. When the pressure from behind can no longer force the blood through the contracted orifice, the murmur disappears.

The friction of opposing currents of blood is often communicated to the surface as a distinct diastolic fremitus, the *frémissement cataire* which is not only audible but may be also palpable in cases in which the action of the heart is strong. The fremitus is, of course, presystolic or diastolic. It is a sign of very great value in diagnosis, especially in cases where the hearing of the observer is not so acute in consequence of aural catarrh or of advancing age. But it is astonishing how much more conception avails than perception, how much more valuable is education of the brain cells than mere acuity of hearing—to know what is heard than to hear.

When the mitral stenosis is pure, that is, when there is no insufficiency, the systole is usually remarkably sharp. The ventricle under these circumstances suffers no dilatation and may contract with greater force upon its diminished contents, and all the more from the fact that the ventricle sometimes shows some degree of hypertrophy.

When, in the course of time, the hypertrophy of the right ventricle begins to flag, dilatation sets in, and there is established sooner or later a relative insufficiency of the tricuspid valve. The pulmonary valve sound at the base, which has hitherto been so distinctly

intensified, now becomes enfeebled and almost inaudible. The presystolic murmur becomes fainter and disappears, while a loud systolic murmur shows itself in the neighborhood of the ensiform cartilage, most distinctly at the fifth intercostal space to the right of the sternum. This murmur depends upon incompetence of the tricuspid valves. Hereupon now ensue all the signs of stasis in the general venous system. The veins pulsate visibly in the neck, the liver is enlarged and sometimes shows pulsations, while stasis in the kidney is shown by the diminution in the quantity of urine, which, besides being scant and heavy, becomes high-colored and albuminous.

The pulse at the wrist varies according to the character of the lesion, and more especially according to the condition of the heart

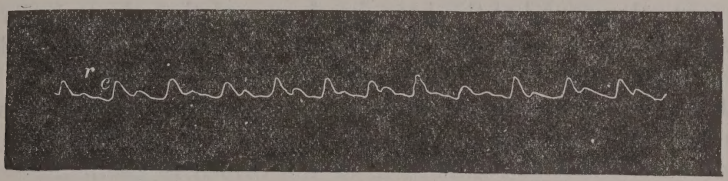


FIG. 8.—Pulse Curve in Mitral Stenosis with Broken Compensation: feeble ascent, feeble force; *e*, elevation; *r*, recoil.

muscle. During the period of compensation the sphygmograph shows a vertical ascent of but little height and diminished amplitude in correspondence with the diminished amount of blood forced into the aorta. The summit of the curve is lightly rounded. The descent is oblique and but little dicrotic. When the hypertrophy begins to give way the heart shows arhythmia, intermissions, and irregularities which disorder the regular tracings of mitral stenosis. The pulse shows arhythmia more marked than in any other valve lesion.

Popoff records a case of a combined lesion (stenosis and insufficiency) with broken compensation, in which the pulse in the right radial artery disappeared at times, probably on account of pressure upon the arch of the aorta by the right auricle and the great venous trunks.

Diagnosis.

The diagnosis of mitral stenosis is usually the recognition of a lesion superimposed upon mitral insufficiency. The patient will have usually shown the signs of insufficiency first and for a certain length of time, when some intercurrent malady, as a new attack of rheumatism or other infection, leads to a new examination of the heart and a recognition of the complication. In quite exceptional cases the mitral stenosis exists alone. The diagnosis is here established

by the relation of the original malady, usually in the nature of an infection, especially rheumatism; by the age of the patient, usually in younger life; by the early appearance of the various evidences of heart failure and stasis, early fatigue, anæmia, cough, dyspnœa, œdema, etc. The pulse is usually small, is often insufficient, and later arhythmic. It becomes much reduced in force, and sometimes almost fades away when the arm is lifted at right angles to the body.

The physical signs upon which the diagnosis really rests are extensive visible pulsation, dislocation of the apex to the left, sometimes as far as the axillary line; increase in the area of dulness, especially to the right of the sternum; the perception by palpation of fremitus, the existence of a presystolic or diastolic murmur heard with greatest intensity at the apex with accentuation of the pulmonary valve sound at the base, or closure of the valves at the base at such intervals as to split or duplicate the second sound. Sometimes the murmur of stenosis is so extraordinarily faint that it may be heard only under increased action of the heart. Sometimes again stenosis exists without any murmur. Strange to say, these are the cases in which the contraction of the orifice is particularly pronounced. It is assumed that the quantity of blood which traverses the extremely contracted orifice is too small to produce a murmur. Minot mentioned a case of mitral stenosis, in which notwithstanding contraction of the orifice to the size of a lead pencil, the presystolic murmur was heard only once and then temporarily. At all other times there were either no auscultatory phenomena or only a short systolic murmur. Potain dwells upon the fact that the diagnosis of mitral stenosis must often be made with the aid of but few of the physical signs. In fact, a strong accentuation of the second pulmonary sound, in the absence of other cause, may suffice to establish the diagnosis.

Of all these signs the most valuable is the presystolic murmur, which, if distinct, actually decides the case; and next the palpable fremitus which is especially pronounced previous to the systole and which ceases suddenly when the apex strikes the wall of the chest. Thus an absolute diagnosis may sometimes be made by palpation alone. But in many cases, as stated, the diagnosis is extremely difficult. The murmur may be absent, the signs of insufficiency may predominate, or all the sounds may be lost under delirium cordis. In these cases a diagnosis may be impossible.

As a rule the diagnosis necessitates the recognition of both insufficiency and stenosis. In such cases, along with the general signs of hypertrophy which belong to both conditions, the murmur, which is double, systolic and presystolic (or diastolic), makes the diagnosis.

Prognosis.

The course of mitral stenosis is always more rapid than that of mitral insufficiency. There is not the same period of latency of symptoms, and this precipitation of signs, which belongs to the nature of the lesion itself, is all the more pronounced because of the coincident insufficiency. Degenerative changes set in much sooner, therefore, in the course of mitral stenosis, and the signs of venous stasis sooner become pronounced. Complications on the part of the lungs show themselves especially frequently and early. Attacks of dyspnœa supervene readily upon effort or emotional disturbance and occur later spontaneously under the distressing signs of cardiac asthma. The effect of the extreme stasis is to interfere with the nutrition of the lungs, and lead to hyperplasia of the connective tissue. Under these conditions hemorrhagic exudations, with pigmentations, develop to constitute the so-called brown induration of the lung, a condition which is so characteristic as to enable the anatomist when he sees it to predict the character of the lesion in the heart. The abnormal relations in the pulmonary artery lead also early to arteriosclerosis, which is usually developed first in the aorta and its branches. All the incidents of valve lesion connected with insufficient blood supply, with venous stasis and embolism, are of more frequent occurrence. The stage of perfect compensation is shorter, so that dilatation of the right ventricle occurs sooner and reaches a higher grade, and thus sooner leads to relative insufficiency of the tricuspid valve. Œdema shows itself sooner about the feet and in less time assumes the proportions of anasarca.

The prognosis is greatly aggravated by the anæmia which sets in in these cases. Young women especially show the aspect of chlorosis. Pregnancy imposes additional dangers. These are the cases especially in which injunction should be put upon matrimony.

Any intercurrent malady is attended by distinctly greater danger, inasmuch as the reserve power of the heart is sooner used up.

The prognosis in an individual case may be determined by the effect of exercise and of digitalis upon the action of the heart and the pulse. The accidental murmurs of heart weakness may be dissociated in this way, as slight bodily movements increase the dyspnœa, arhythmia, etc., in organic disease, but improve the condition in mere muscular weakness.

The *treatment* requires an earlier and more careful consideration of prophylaxis that the heart muscle may be conserved as long as possible, an earlier resort to the heart stimulants with especial caution as to their abuse, and systematic development of the heart muscle

under cautiously graded exercise. Rapid and tumultuous action should be subdued. It is easy to understand why the left ventricle may be better filled under a longer duration of the diastole so that a diminished frequency in the action of the heart is decidedly favorable to compensation.

Aortic Insufficiency.

Insufficiency of the aortic valves belongs among the earliest recognized affections of the valves. The condition was noticed by Morgagni and Sénac but seemed to have passed from recognition until our knowledge of it was revived by the observations of Hope and Corrigan in the third decade of the nineteenth century. The effect of insufficiency at the aortic valve is so great and the phenomena which immediately attend it are so striking as to have directed to this lesion more attention than to any other.

Insufficiency of the aortic valve implies imperfect closure under the reflux of blood, so that a certain quantity of blood regurgitates into the left ventricle. The valves become incompetent through two distinct processes, to wit, endocarditis and arteriosclerosis or atheroma. Moreover, the aorta may itself become so dilated as to render the valves relatively incompetent. This condition is observed more especially in connection with aneurysm of the aorta, which, when it occurs in the ascending portion, involves in its spindle-shaped dilatation the orifice of the aorta in the region of the valves.

Dombrowski is not willing to admit any dilatation of the left auriculo-ventricular (mitral) orifice, because the orifice is surrounded with a tendinous ring that is but little distensible. Any dilatation of this kind here is the fault of the valve whose flaps fail to close the orifice on account of muscular insufficiency. But in the case of the aorta the condition is different. The aorta itself does give way and the ring of insertion of the valves does dilate. This dilatation is much more serious in the case of the aorta, as it means arteriosclerosis and is permanent and progressive, while the relative (functional) insufficiency of the mitral valve may be remedied by simply toning up the heart muscle.

Fenestration of the valve is seldom the cause of insufficiency. Trauma may play a more important rôle. Thus Louis reported a case of rupture of one of the aortic valves during severe muscular strain, and Greenhow a case of incompetency of the aortic valves, produced by a strain, with subsequent endocarditis and the formation of a polypoid body hanging into the left ventricle and causing ulceration of the ventricular septum and erosion of the adjacent cusp of the

valve. A rare case was described by Fütterer in which rupture of the wall of the aorta had developed an adventitious sac, a kind of false pocket, which was interposed between the intact aortic valves and thus interfered with their function.

The process of arteriosclerosis or atheroma occurs more particularly in advanced life, so that insufficiency of the aortic valves, the result of atheromatous change, is more frequent in old age. Typical atheroma may occur, however, in earlier life as the changes of age are precipitated, especially by alcohol, syphilis, or hard work. The first of these factors is also the most prominent in the production of aneurysm, so that the spindle-shaped dilatation of the aorta which produces relative insufficiency occurs frequently in younger life and persists at maturity. The second factor, syphilis, is prominent in the production of tabes dorsalis, and Rosenbach calls attention to the frequent coincidence of aortic insufficiency with tabes dorsalis on account of the associate arteriosclerotic changes in the aorta. Endocarditis, being the expression of an infectious process, belongs to earlier life.

As a rule, the endocarditic process begins with the mitral valves and extends, usually in some subsequent attack of rheumatism or other affection, to involve the aortic valves. Here the process may be either benign or malign. As the malignant (septic) forms more commonly terminate fatally, the sclerotic changes which are observed are usually sequels of simple (benign) endocarditis.

The degree of incompetency varies greatly. Sometimes the little finger can be introduced through the valves. On the other hand, rupture of a valve or processes of thickening may leave only a buttonhole slit. The condition most frequently encountered is a triangular opening from defective closure on account of thickening and shrinkage along the free borders of the valves. Through this orifice the blood falls with the force of gravity, and is forced under the strong resilience of the aorta back into the ventricle whence it was discharged.

This reflux of blood soon distends the left ventricle and leads to dilatation of the cavity. The walls of the left ventricle immediately begin to undergo hypertrophy and in this way overcome the defect. As the hypertrophy in this case concerns the left ventricle, and as the left ventricle is naturally a powerful muscle, the hypertrophy becomes extreme. Then as so much of the blood falls back into the ventricle, the aorta is comparatively but little filled and the tissues suffer from want of nutritive material. The tissues make this want known through the nervous system and the heart is thus stimulated to its utmost effort to supply the demands. It is, therefore, in these

cases of aortic insufficiency that the hypertrophy assumes at times proportions which may be called colossal. The heart of man approximates in magnitude that of the ox, and in fact this heart is distinguished as the cor bovinum.

The peculiar lines and opacities so often seen in the endocardium of the ventricular wall below the valves show the resistance which is offered by the muscular tissue in this region. The extreme enlargement of the left ventricle under this hypertrophy increases the size of the heart, especially to the left, so that the organ assumes a more

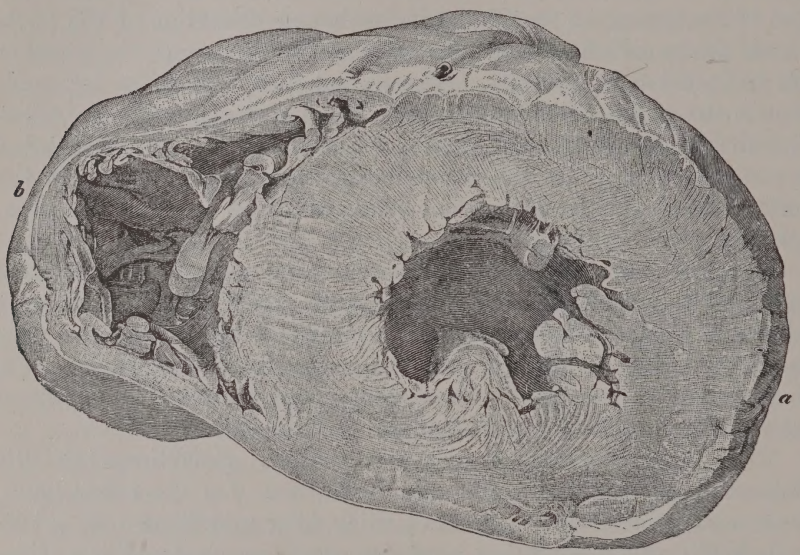


FIG. 9.—Hypertrophy of the Left Ventricle in Consequence of Insufficiency and Stenosis of the Aortic Valves. *a*, Left ventricle; *b*, right ventricle. Cross-section; natural size. (Ziegler.)

vertical position in the chest. The outlines of the whole heart may be traced at times beyond the mammillary line, and the apex may be found as far to the left as the axillary line.

The force of the reflux of blood is indicated, not only by the dilatation and hypertrophy which take place to secure its final expulsion, but also by the peculiar and characteristic flattening of the trabeculæ in the cavity of the ventricle. The flattening of the trabeculæ and papillary muscles, which occurs in consequence of the pressure, is seen, according to Schwalbe, most pronounced on the posterior wall of the heart.

The dilatation of the left ventricle is observed first at the pars arterialis under the pressure of the regurgitating blood. Hypertrophy develops with it nearly simultaneously. The hypertrophy of

the left ventricle compensates the lesion, when it may force a sufficient quantity of blood into the aorta to make up for the loss of the refluent blood, that is, when it may pick up this additional load with its own natural contents and discharge it to the body.

But it is the work of Sisyphus which is thus thrown upon the left ventricle, in that it must unload itself of a burden which continuously falls back upon it.

The right ventricle is either normal or shows moderate dilatation and hypertrophy. These conditions result as the consequence of insufficient work upon the part of the left ventricle, which leads to stasis in the left auricle, and this stasis communicates itself through the lungs to the right ventricle which must then undergo dilatation and hypertrophy.

Morbid Anatomy.

The nature of the lesions depends upon the character of the inflammatory or degenerative process. Ordinarily the valves are found thickened and more or less adherent. Sometimes a segment or part

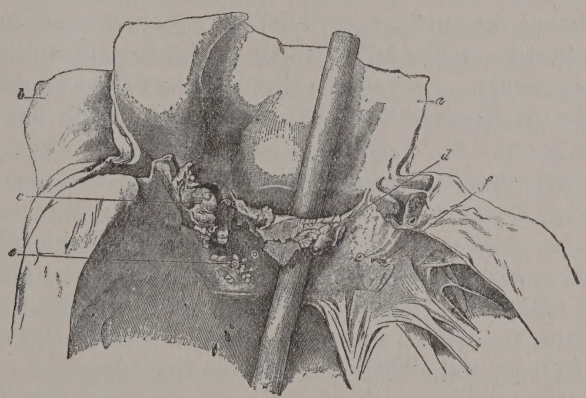


FIG. 10.—Ulcerative Endocarditis with Perforation of the Aortic Valves. *a*, Aorta; *b*, pulmonary artery; *c*, thrombotic deposit upon a segment of the valve; *d*, perforation; *e*, ulcer of the septum; *f*, ulceration of the mitral valve.

of a segment is agglutinated to the wall of the aorta. In other cases the valve is perforated in consequence of the rupture of an aneurysm in its structure or of a final fatal rupture under progressive degenerative processes. Sometimes a segment or part of a segment is torn from its base. This accident results in a sudden insufficiency. The more minute openings or fenestrations, which are not infrequently found along the edge of the valve, produce no incompetency as they lie beyond the line of closure.

Under the atheromatous process the valves are thickened, ren-

dered opaque, and show grayish-yellow patches of hyaline or atheromatous change. Sometimes the necrotic process breaks the surface or produces a more extensive solution of continuity in the structure of the valve, which terminates most frequently in the production of the atheromatous ulcer or which may result in perforation.

Not infrequently these processes coincide. In this case the lesion begins with an endocarditis with its characteristic thickening and deposit of fibrin, to constitute excrescences or verrucosities like those found on the mitral valve. Atheromatous degeneration may now set in later as a degeneration in the valves themselves or as an extension from the process which began in the aorta. The common lesion is a thickening and shrinkage from adherence of the segments to each other with rolled and thickened borders. In these cases the finger may be often inserted behind the valve, whose texture is then seen to have lost its translucency and is felt to have lost is elasticity and pliability.

Coarse degrees of insufficiency may be detected by the hydraulic test, that is, by simply pouring water into the aorta before the heart has been laid open or the base of the aorta has been incised. In this experiment the aorta should be cut square across, and should be held so that the water falls directly at right angles to the plane of the three segments. Any escape through the valves would be recognized by the discharge of water through a slit in the left ventricle. The valves should be considered insufficient only when the water escapes in this way in considerable quantity, as the penetration of a few drops of water would count for nothing. In all cases the mere pouring in of water would fail to show the action of the valves under the conditions which take place in the circulation where the pressure is equal to that of a column of water of the size of the aorta three metres in length. Such a pressure naturally unfolds the valves with considerable force and closely apposes the free edges.

Drasche found the width of the aortic valves under tension to vary between 33 and 34 mm., the depth of the pockets between 15 and 17 mm., in an average circumference of the aorta. It must be remarked again, however, that any mere measurement of the surface does not furnish an absolute indication of the relation of the valves to the orifice, as it omits the important factor of contractility of muscular tissue which regulates the size of the orifice and which cannot be estimated in the dead body.

It is not probable that the aortic valves lie flat against the wall of the aorta. The length of the free border of a segment is longer than the arc of the aorta which it subtends, so that the valves stand out somewhat like pockets. In experiments with animals a sound which

is introduced into the aorta usually finds its way directly into one of the sinuses or pockets of the valves, and it is almost impossible to introduce the sound into the ventricle without injuring one of the valves.

Symptoms.

Aortic insufficiency may remain latent for a long time and the patient may show no sign of heart disease until some intercurrent malady, extraordinary demand, or sustained depressing emotion may exhaust the reserve force of the heart and lead to palpitation, pain, or other sign of nervous or organic distress. Aortic insufficiency is often introduced insidiously. In individuals who lead sedentary lives, more especially under intellectual stimulus, and who begin to show signs of nervousness with distress in the region of the heart, palpitation, vertigo, etc., a careful examination of the heart will sometimes disclose typical insufficiency of the aortic valves.

On the other hand, the symptoms in a few cases set in suddenly. Thus Bellotti reports the sudden development of an aortic insufficiency in consequence of extraordinary strain in the cases of two men, aged respectively twenty-six and forty years. The condition was revealed by precordial pain, intense dyspnœa, and a musical diastolic murmur beside the attenuated second aortic sound. Other signs were absent. Entire recovery occurred under rest. The lesion was interpreted as a rupture of a valve segment with fenestration, a condition which admits of perfect restoration.

So soon as the hypertrophy begins to flag in its force the symptoms of heart failure begin to appear. As the brain is exceedingly sensitive to alterations in the blood supply, persons affected with aortic insufficiency suffer from vertigo, headache, and insomnia. Patients are liable to be tormented with throbbing sensations from pulsation of the carotids in the neck with a feeling of fulness in the head, with humming sounds in the ears, with sparks before the eyes. Cerebral hemorrhage is not uncommon in consequence of arteriosclerosis or brain embolism. Insufficiency of the aortic valves is often accompanied also with disease of the kidneys. The face is usually pale in contrast with the duskiness and ectasia of the vessels in later stages of mitral disease.

As many cases are associated with the changes of arteriosclerosis and atheroma, cases of aortic insufficiency are especially liable to be attended with pain, which varies in every degree of intensity from simple discomfort and distress to the indescribable agonies of angina pectoris.

The hypertrophy which takes place in the right ventricle postpones for a long time symptoms on the part of the lungs. Under any ex-

traordinary demand this hypertrophy begins to give way and patients suffer from attacks of dyspnœa, at first under exercise, later paroxysmally and without provocation. As aortic insufficiency depends often upon arteriosclerosis, a widespread process in the body, it is not surprising to learn that this lesion may be found in association with changes in other organs, in the brain, kidneys, and in the body of the heart itself. Aortic insufficiency is found frequently in connection with atheroma of the vessels, of the vessels of the brain, with Bright's disease and with sclerosis of the aorta and coronary arteries. Attacks of pseudo-angina and real angina and attacks of cardiac asthma occur naturally in the course of this condition.

Physical Signs.—The great enlargement of the heart may be seen at times on inspection of the surface. The front of the thorax may be bulged outward. This condition is noticed of course only in the young, in whom the wall of the chest is distensible. The great force of the heart is observed in the increased impact which may shake with violence the wall of the thorax. Patients sometimes complain of the powerful throbbing of the heart.

The impact of the heart is so diffuse that it is not only seen in the region of the heart, but extends over the whole left side of the chest. Sometimes it shocks the whole chest, and in extreme cases actually agitates the whole body of the patient and even the bed upon which he lies.

The thorax not infrequently shows at the region of the apex systolic retractions, which involve usually only the intercostal spaces but may, in yielding chests, involve also the ribs. These retractions have nothing to do with the systolic sinkings of concretio pericardii, but are due solely to the fact that the lung is retracted so far to the left under the increased volume of the heart that it may no longer interpose itself to cover the heart sufficiently during the systole; the systolic aspiration therefore retracts the wall of the chest.

Palpation renders these phenomena even more distinct. The heart is felt to bound under the hand and the impact is sometimes so great as to cause the student who is making his first observations to remove the ear from the surface of the chest. The area of the apex beat covers a wider surface, over all of which the heart beats with intensity. The apex strikes, instead of in the fifth, in the sixth, seventh, or even in the eighth intercostal space, and sometimes as far to the left as the axillary line itself. Sometimes there may be felt a distinct vibratory thrill, though the fremitus is by no means as common as in the case of stenosis of the aortic valves. But not at all infrequently the conditions coincide; there is both insufficiency and stenosis.

Percussion reveals the increase in the outlines of the heart. The dulness extends from the right border of the sternum to the mammillary line, and from the second to the sixth ribs.

When the left ventricle first begins to flag in its force so that blood accumulates in the cavity, extra work is thrown upon the left auricle to discharge itself of its contents into the left ventricle. The left auricle, however, is little fitted for extra work. It soon yields under the strain and suffers a certain degree of distention. As the blood now accumulates in the left auricle the pulmonary veins may no longer discharge their contents with the same ease and rapidity and blood is dammed back into the lungs, to produce ectasia of the capillaries and to lead to increase of pressure in the pulmonary artery. So soon as the blood pressure increases in the pulmonary artery the right ventricle must undergo hypertrophy. Under this hypertrophy the right ventricle is able nevertheless to empty itself and to force the blood through the distended capillaries into the pulmonary veins and left auricle. The right ventricle takes, therefore, a direct part in the compensatory process, and it is this enlargement of both ventricles which gives to the heart its great size. Moreover, this universal hypertrophy contributes in large degree to make the prognosis favorable in a condition which would otherwise be extremely dangerous. It will readily be seen, therefore, that the process which develops slowly furnishes a much more favorable outlook than those which develop rapidly, as the ventricles have under a slow process time for gradual, and finally, perhaps, entirely compensatory hypertrophy.

As an absolute fact the hypertrophy of the left ventricle becomes so extreme as to be able in the course of time to dispense with the assistance of the right ventricle. So soon as the ventricle is able to empty itself of blood the pressure in the left auricle is relieved. There is then no longer stasis in the pulmonary veins and lungs and increase of pressure in the pulmonary artery. The hypertrophy of the right ventricle may therefore subside to large degree and may not renew itself until the left ventricle begins to flag.

The reflux of blood through the narrow orifice into the enlarged cavity of the left ventricle produces a murmur which is heard in greatest intensity over the site of the aortic valves, viz., in the second interspace to the right of the sternum, and which corresponds in time with the dilatation of the ventricle, that is, with the period of the diastole. The murmur is, therefore, synchronous with the second sound of the heart, whose place it often takes or to which it is appended. Bernheim hears the murmur a little before the second sound and pronounces it prediastolic. The reason of it, he says,

is simple; the blood rushes back before the rigid valves find time to close.

The murmur is propagated along the right border of the sternum or obliquely toward the apex of the heart behind the body of the sternum in the direction of the refluent blood (Sée), but is sometimes transmitted directly across the sternum to the left side where it may be heard distinctly in the third interspace (Petit). Where the murmur is loud it may be heard in wide dissemination, sometimes, indeed, even at a distance from the body, and there are cases in which the murmur is so intense as to disturb the rest of the patient. As a rule, however, the murmur is feeble and is confined to the point of closest proximity of the valves to the wall of the chest, that is, to the third interspace close to the edge of the sternum or at the junction of the third costal cartilage with the sternum. The murmur is always most intense at first and gradually fades away, as the main amount of blood falls at once by gravity and under compression of the aortic walls through the incompetent valves. The murmur has a tolerably constant character; it is soft, gushing, or like the sound of "swish," is long drawn and is usually of no great intensity.

In rare cases the murmur may be rough, sawing, or have a musical tone. In these cases it may sometimes be heard at some distance from the patient.

The intensity of the murmur stands in some relation with the size of the opening. When the opening is small the murmur is faint or may fail entirely. On the other hand, when the opening is so large as to approximate the normal orifice, the murmur diminishes in intensity. Deposits may make the murmur rough. Great pressure in the aorta intensifies it and lessened pressure diminishes it. Alterations in the posture of the patient have also a certain effect (Kovacs).

Sometimes the lesion is present and there is no murmur. This may be the case in recent processes, in the presence of certain complicating valve lesions, in extreme stenosis, and in mitral affections. The murmur often ceases for considerable time in consequence of great losses of blood. According to Leube the absence of murmur is noticed most frequently when the insufficiency reaches the highest grade, so that when water is poured into the aorta at the autopsy it flows through in volume. This degree of insufficiency may be produced by extreme shrinkage, so that only relics of the valve remain. With such a lesion there is no friction from whirling, and thus no condition for the development of a murmur. The second sound, which is heard at the orifice of the aorta in these cases, is the

propagated sound from the closure of the valves of the pulmonary artery.

The murmur may be absent also in the beginning of aortic insufficiency, while the other symptoms, for instance the pulsation of the spleen, may be very distinct. The absence of the murmur in this case is explained by a lessening of the blood pressure and of the rapidity of the circulation in consequence of degeneration of the heart muscle. Drasche and Litten have reported cases in which the murmur was intermittent.

Insufficiency of the aortic valves is, in typical cases, always accompanied by a systolic murmur at the apex on account of a simultaneous incompetency of the mitral valve. Fisher finds the explanation of the presystolic apex murmur of aortic insufficiency in implication of the papillary muscles and their tendons, or in relative incompetency of the mitral valve unsatisfactory and would rather attribute it to some disturbance in the nervous mechanism. But this explanation is itself too vague to be satisfactory.

Accentuation of the second pulmonary sound occurs only in the stage of disturbed compensation with the establishment of relative insufficiency of the mitral valve. This accentuation is thereby a sign of stasis in the pulmonary circulation.

Attention has been called by a number of observers, especially in our own country, Flint, Guitéras, and others, to a presystolic murmur which may be sometimes appreciated at the base of the heart. This murmur has met with different interpretations. When distinct or more decidedly synchronous with the systole, it has been considered evidence of a coincident mitral stenosis. Potain considered it as a muscular sound due to the hypertrophy of the left ventricle. The fact that this murmur is sometimes attended with fremitus dissociates it from anything like an extra-cardial, for instance a cardio-pulmonary, bruit. In their further studies Sampson and Potain locate the bruit in the mitral valve, whose larger segment, crowded back by the refluent blood, makes a kind of relative insufficiency of the mitral valve. The irregular vibration of this valve, interposed as it is between the refluent blood from the aorta and the effluent blood from the ventricle, produces the fremitus and the murmur.

The murmur of aortic insufficiency is usually a blowing sound of such intensity as to be disseminated over a considerable portion of the chest and to be propagated into the carotid arteries. It varies, however, as stated, in every degree of intensity and character, is sometimes soft as a whispered *who*, at other times loud and rough. In association with the enlargement of the heart this murmur is characteristic of the condition. It ceases only when the hypertrophy

begins to give way and the action of the heart becomes feeble and irregular.

Alterations of the Pulse.

The sudden distention and collapse of the arteries which results from the sudden filling and instantaneous reflux of blood through the incompetent valves makes itself manifest in a peculiar alteration of the pulse—that is, the blood wave is felt in the radical artery almost at the very moment of systole as the artery is distended under the powerful contraction of the hypertrophied ventricle, and is then lost to the touch as the artery almost instantly empties itself in both directions, forward into the capillaries and backward into the heart. This sudden filling and collapse of the artery was first of all noticed and described by Corrigan, of Dublin, in 1830. This distinguished physician, though he had but six beds under his care, made such accurate investigations and clear descriptions of the condition as to have connected his name both with the disease and the pulse, for aortic insufficiency often goes by the name of Corrigan's disease, and the blood wave which appears and disappears in a flash is known

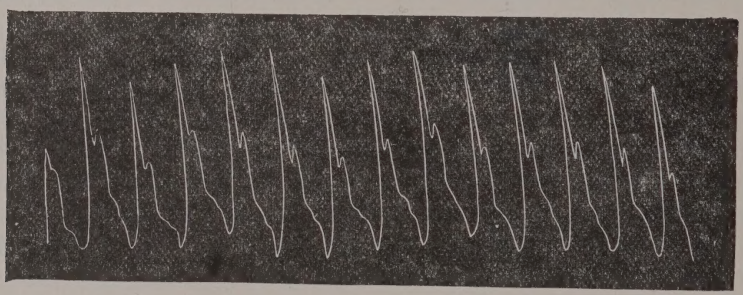


Fig. 11.—Pulse Curve in Aortic Regurgitation. High perpendicular ascent; sudden descent. Cannon-ball pulse.

as the Corrigan pulse; "cannon-ball" pulse and "pistol-shot" pulse are common equivalents which suggest the characteristic conditions. Sée called it, on account of its rapidity, the pulsus celer, because the duration of it is so much less than that of the normal pulse.

Petit observes the fact that the condition is much more marked when the arm is lifted from the body in a position which favors the more immediate reflux of blood.

The sphygmographic trace shows in these conditions a vertical ascent of considerable height, always greater than normal, with such sudden collapse as to make of the apex an extremely acute angle. The line of descent is also more abrupt and more distinctly dicrotic,

as indicative of the feeble arterial tension. These characteristics have been utilized to establish a diagnosis, but since it has been found that the same abrupt elevation, acute angle at the summit, and zigzag decline may be observed in other conditions, as in aneurysm of the aorta and certain cases of anæmia, the sphygmograph may furnish only corroborative but not pathognomonic evidence. Moreover, these conditions are only fully present under some degree of hypertrophy of the left ventricle.

The pulse of Corrigan is visible also in the great vessels in the neck, in the carotid and subclavian arteries. The French speak of the rhythmical undulation as a "*danse des artères.*"

The powerful action of the heart propels the blood with such force that the pulsation of the arch of the aorta may be perceived in the jugulum. The carotids and the temporal arteries pulsate with force to show alternate distention and collapse, and the smaller arteries, which are usually invisible, are now seen to pulsate distinctly.

Müller found a rhythmic pulsatory movement and swelling of the veil of the palate, of the uvula, and of the tonsils, as well as a rhythmic increase in the redness of the palate. Merklen also saw the pulse of the soft palate, described by Müller, in a case of aortic insufficiency occurring in a man twenty-one years of age; the rhythmic swelling of the uvula was especially distinct in this case.

This capillary pulse, to which attention was first called by Quincke, may be made visible by producing artificial hyperæmia in the skin. For instance, if the forehead of the patient be rubbed with a cloth the hyperæmic surface thus produced becomes redder under the systole and the flush is increased at the borders. The systolic flushing of the nails is observed, especially after light pressure. The flush of the systole encroaches upon the white at the root of the nail.

Balfour is not willing to accord any diagnostic value to the capillary pulse. Certain authors declare that it is never found in health, that it occurs only in heart diseases, and in them in insufficiency of the aortic valves in more than eighty-three per cent. of cases. It is certainly frequent in this condition but is by no means pathognomonic of any particular form of heart disease. Rosenbach declares that he never saw this phenomenon distinctly.

Quincke also called attention to the centripetal venous pulse in this condition. The force of the heart propels the blood completely through the domain of the capillaries into the peripheral veins. The condition implies not only a strong heart and a rapid pulse, but also a relaxation of the arteries and veins. This venous pulse is seen best in the hand and arm. Like the capillary pulse it is not pathog-

nomonic of insufficiency but is found also in anæmia and fever. The capillary pulse has no absolute value, as the same pulse has been observed in the hypertrophy of the heart which results from arteriosclerosis, Bright's disease, lead-poisoning, etc. The capillary pulse is really an indication of the force of the left ventricle which is strong enough to propel the blood wave through the whole arterial system and directly into the capillaries. The condition is most distinctly observed in aortic insufficiency because of the contrast which is offered in the sudden distention and sudden emptying of the vessels under the double escape behind as well as before.

Corrigan called attention also to a fremitus which may be perceived in the arteries, as the expression of the friction of the rapid current, and Traube observed the double sound which results from irregular vibration of the arterial walls. The first sound results from the distention of the artery under the inflowing wave of blood, the second feebler sound results from the rebound of the artery after it has been stretched, constituting the dicrotism which is perceptible to the touch and is more distinctly manifest in the sphygmogram. This double sound is best appreciated in the larger arteries, as in the femoral, and in the study of it all compression of the artery by the stethoscope must be avoided.

Duroziez developed a double murmur in these cases in the larger arteries, especially in the femoral artery, under strong compression of the stethoscope. This murmur was not only double but intermittent, and to this "double souffle intermittent" were ascribed pathognomonic properties.

The first murmur results from compression of the arterial trunk and may be developed in the same way in any artery in a state of health. The second murmur is due to the rapid reflux of blood, and this murmur is found only in aortic insufficiency. Potain is unwilling to ascribe this murmur simply to the retrograde current of blood, but attributes it likewise to the compression of the stethoscope. As this double souffle has been observed also under the same conditions as the double sound, it has no real pathognomonic value. The spontaneous tone in the crural artery under a gentle pressure of the stethoscope is, according to Hochhaus, rare and not characteristic. It occurs always in insufficiency of the aortic valves, and almost always in compensated mitral insufficiency, in anæmia, and in febrile disease. Matterstock says that the double sound is found most frequently in insufficiency of the aortic valves, fever, and lead-poisoning. It may be heard also during pregnancy.

The truth is, the double sound in the crural arteries is heard in health only under increased activity of the heart, as, for instance in

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running and jumping, but is heard in aortic insufficiency under light pressure below Poupart's ligament. At least there is developed in this way a very loud clear diastolic tone.

Both the double sound and the double murmur may be heard at times in the subclavian and axillary as well as in the crural artery.

Diagnosis.

In the presence of the great hypertrophy of the left ventricle which distinguishes this condition and during the existence of the murmur, however faint, the diagnosis is not difficult. The genetic relationship is more extensive than in mitral disease, as it includes atheromatous change and the diseases which produce arteriosclerosis. Thus a previous history of alcoholism or of syphilis would shed more light upon disease of the aortic valves than upon affection of the mitral valves. The previous history of some acute infection is not so essential in the diagnosis of disease of the aortic valves.

Endocarditis usually begins in the mitral valves and extends so as to involve the aortic valves in some subsequent attack of the original malady, as of rheumatism, but in the more exceptional case endocarditis may begin in the aortic valves and be confined to this region for a time. Insufficiency of the aortic valves occurs most frequently in youth and middle life, not so much because of the frequency of endocarditis at this time but because of the frequency with which atheromatous change is precipitated by alcohol, syphilis, saturnism, gout, diabetes, etc.

When all the physical signs are present no lesion of the heart is more readily recognized, for there is no affection which leads to such enormous hypertrophy with corresponding increase of force in the action of the heart. Moreover, a diastolic bruit points always more definitely to organic affection than a systolic bruit which may depend upon mere poverty of the blood, more irregular vibrations, or feeble muscular contractions.

The increased hypertrophy makes itself manifest by the great increase in dulness above and to the right and left, and by the extreme reaching over of the apex to the left, even as far as the axillary line. The bruit is heard in greatest intensity in the third interspace to the left of the sternum and synchronous with the second sound of the heart. It varies in every degree of intensity but is usually faint, and only exceptionally intense or loud. Faint as it is, however, it may be propagated into the carotids. In rare cases the presence of a diastolic murmur may be accidental. It may then have its origin in a vein near the heart. Such a condition develops especially in the anæmic. The murmur is developed either from the inferior vena

cava, when it may be heard especially loud at the lower part of the sternum, or in the jugular vein, when it is heard in intensity in the neighborhood of the clavicle. These conducted murmurs may be easily recognized by the fact that in the case of the jugular vein they disappear upon pressure on the vein (Leube).

Extremely valuable evidence is furnished by the pulse. The arteries are subjected to sudden distention and collapse, constituting the "pistol shot" pulse, and the force is so great as to propagate the pulse into the capillaries. Finally the double sound, which is produced by sudden distention and collapse, may be heard in any of the great arteries, and the double souffle of Duroziez and double sound of Traube, which result from pressure with the stethoscope, may be distinctly heard, especially in the femoral artery.

The diagnosis is thus easy in the height of the disease and when the lesion is largely compensated by the hypertrophy of both ventricles. Sometimes the diagnosis may be read at once by a glance at the sphygmogram.

But the diagnosis is by no means always so easy. Sometimes the lesion is unattended by a bruit. Affections of other valves or other affections of the same valve disturb the phenomena. In the later course of the disease, when hypertropy gives way and dilatation ensues, it may be absolutely impossible to distinguish the special lesion. Huchard dwells upon the difference which he has observed in the symptomatology of an aortic insufficiency produced by rheumatic endocarditis and arteriosclerosis. In endocarditis the valve lesion dominates, while in arteriosclerosis the valve lesion is secondary. "Arterial" aortic insufficiency is distinguished by an unsually loud, rasping, often musical ("piaulant") diastolic murmur, by defective hypertrophy of the left ventricle with weak apex stroke, with a tendency to dilatation. Usually there is dulness at the region of the manubrium sterni from dilatation of the arch of the aorta. Not infrequently there are attacks of angina pectoris. The pulse of arteriosclerosis is characteristic. Then there are other symptoms of disturbance of circulation in the brain, liver, and kidneys, *e.g.*, vertigo, dyspnœa, which originate neither in the heart nor in stasis in the lungs, but are of toxic nature, dependent often upon defective activity of the kidneys (even without albuminuria) so that heart tonics have no effect, while the condition is often improved by a vegetable and milk diet.

Relative insufficiency occurs in consequence of an advanced arteriosclerosis. The development of it is therefore in contrast with the organic disease, always latent, slow, and insidious. Along with palpitation, vertigo, dyspnœa, develop gradually signs of the dilated

arch of the aorta, to wit, pulsation in the jugulum, dulness at the sternum, metallic second aortic sound. Finally, there may be found in the diastolic murmur the proof of the unfortunately irreparable, relative insufficiency of the valve. The pulse is hard, tense, and rapid, and the sphygmograph shows the ascending line more perpendicular than in the pulse of pure aortic insufficiency (Dombrowsky). All the other symptoms are the same in both conditions.

Relative insufficiency of the aortic valves is, as stated, rare, but occurs under excessive dilatation of the aorta at its orifice. This relative insufficiency of the aortic valves is distinguished, according to Groedel, by a diastolic musical murmur, which is unusually loud. The murmur is produced by the irregular vibration of the valves, which are affected in their structure during the passage of the regurgitating blood. Now, while it is true that a singing diastolic murmur may occur from other causes, especially from perforation of the pockets of the semilunar valves, and may occur also temporarily in health, yet when the musical note is distinctly present it should always excite the suspicion first of relative insufficiency of the aortic valves. The diagnosis of this relative insufficiency may be supported by the demonstration, by means of percussion, of dilatation at the orifice of the aorta (Leube).

Any lesion of the aortic valves should be distinguished from affection of the mitral valves by the fact that the murmur in the course of the aortic valves is always to be heard in greatest intensity at the base, and lesion of the aortic valves may be separated from a similar lesion of the pulmonary valves, aside from the extreme rarity of pulmonary affection, by the fact that the murmur is heard in greatest intensity in the aortic valves upon the right and in the pulmonary valves upon the left of the sternum. Moreover, the peculiar pulse of aortic insufficiency is absent in any other valve disease.

The diagnosis, however, can never rest upon a condition of the pulse alone, as a similar capillary pulse may sometimes be observed in any condition which leads to marked hypertrophy of the heart, as in Bright's disease or in arteriosclerosis from any cause, or even in any violent action of the heart from nervous cause independent of hypertrophy, as in certain anæmias, in saturnism, and in gout.

Aneurysm of the aorta causes really the chief difficulty in diagnosis. Sometimes, indeed, the conditions coincide, and it is the dilatation of the aorta which makes the valves incompetent. In other cases the diagnosis may be established by the fact that aneurysm affects the arch more frequently than the ascending aorta, so that the bruit and thrill characteristic of aneurysm are perceived above and in the notch as well as at the left of the sternum. Aneu-

rysm of the ascending aorta usually, however, shows the bruit and thrill in greatest intensity to the right of the sternum, but then commonly over a much wider area, extending sometimes two to three inches to the right of the sternum. The dislocation of the whole heart downward and to the left on account of elongation of the aorta in consequence of aneurysm, may closely simulate that increase in all the diameters which occurs in aortic insufficiency. It will be noticed that the bruit and thrill coincide with the systole in aneurysm and with the diastole in aortic insufficiency. The dulness in aneurysm in an advanced case extends over a much wider area than is ever reached even by the big heart of aortic insufficiency. Further, the bruit and thrill of aneurysm are most intense at some distance from the heart and become enfeebled as the stethoscope is made to approach the heart. Sometimes the diagnosis is established by the inequality, as regards both time and volume, shown in the radial pulse on the two sides. Aneurysm never produces the peculiar pulse of aortic insufficiency and never causes the double sound of distention and sudden collapse in the arteries, or the double souffle under pressure with the stethoscope. If these things are observed in connection with aneurysm they furnish evidence of at least relative insufficiency of the aortic valves.

It will be remembered always that insufficiency of the aortic valves has genetic relationship with many and varied conditions, while the great bulk of aneurysms rest upon syphilis, in connection often with strain and alcoholism.

Prognosis.

In a general way the prognosis is favorable, at least for a long time. As is the case in all the lesions of the heart, it is most favorable in the young in whom the nutrition is good and compensation is more or less perfect. Because both ventricles take part in the process of compensation, and because the left ventricle especially is capable of such enormous hypertrophy, insufficiency of the aortic valve is often regarded as the most favorable of all the valvular lesions. But this optimistic view is damaged to considerable extent by the fact that the lesion often depends upon a degeneration which is widespread in the body and which involves the integrity of other organs, the aorta, the kidneys, the brain, etc. Therefore it may not be said that insufficiency of the aortic valves is as favorable as insufficiency of the mitral valves. Insufficiency of the mitral valves is not incompatible with long life. Long life is, however, seldom reached with insufficiency of the aortic valves, though the lesion is longer latent and compensation may last under favorable circumstances for fifteen to twenty years.

It has indeed been asserted that patients may recover entirely from insufficiency of the aortic valves; at least it has been asserted with some degree of proof that all evidence of the lesion has entirely disappeared. Potain, for instance, claimed to have observed the disappearance of the signs of a recent case, and Leyden demonstrated a case in which an insufficiency of traumatic origin had been repaired by a fibrous cicatrix. It is certain that such reparation may take place in animals in which this lesion has been produced. Any shrinkage of the valves may be compensated, as in the case of the mitral valve, by dilatation and extension of still distensible parts, so that the defect may be entirely overcome.

The signs of aortic insufficiency disappear entirely in only the rarest cases. The rule is that the condition persists and that in the course of time the degenerative changes which are always present in the heart muscle in every form of valve lesion get the upper hand, so that the symptoms of dilatation, to wit, arhythmia, tachycardia, and the signs of general heart failure, dyspnœa, vertigo, oliguria, etc., gradually supervene. Under this degenerative process the force of the heart flags, the murmur is no longer to be heard at the base, and the strong pulse is substituted by the weak and irregular pulse of heart failure. The disappearance of the characteristic signs of the condition is, therefore, ominous enough. Martin Durr reported a case of sudden death in aortic insufficiency from œdema of the lungs, an unusual complication in this lesion. The patient, a smith, aged forty-one, was the victim of angina from arteriosclerosis.

Treatment.

The best treatment of aortic insufficiency is that which longest conserves the compensatory hypertrophy of the left ventricle. During the stage of tumultuous action the patient should observe rest. Violent contractions may be subdued by cold compresses or by the application of an ice-bag. Less violent motions may be controlled by large or by moderate doses, grs. xx. to xl., of the bromides, especially the bromide of sodium. The clinician will be cautious with his interference in subduing the action of the heart during this period of tumultuous action, which often represents a stage of adjustment, or of fresh insult to or irritation of the endocardium.

Pain, however, always calls for relief. Mere discomfort or distress may be relieved by the bromides. Sometimes a small dose of phenacetin, grs. v.–x., quickly subdues neuralgic pain. Antipyrin, grs. v., is often more effective and is in this dose quite safe. The salicylates, salol grs. v.–x. every two to four hours, or quinine in similar dose, may secure more permanent relief. Pain which is so

severe as to simulate angina pectoris may call for the use of nitroglycerin, one drop of the $\frac{1}{100}$ of one-per-cent. solution in a teaspoonful of water every half-hour or hour. The pains of angina pectoris may be relieved at once by the inhalation of amyl nitrite.

Attacks of dyspnœa subside usually under the administration of a diffusible heart stimulant, such as the tincture of camphor, the tincture of valerian, ʒ ss.–i. in a teaspoonful of water, or Hoffmann's anodyne in equal dose. Dyspnœa and pain are, as a rule, controlled at once by morphine, especially when given subcutaneously.

Where the condition may be assumed to depend upon arteriosclerosis, whether of specific origin or not, the iodides, especially the sodium iodide, should be administered in small dose, grs. v., three times a day, preferably before meals.

The compensatory hypertrophy is best sustained by systematically graded exercise with abstention from all bad habits, especially the use of agents which irritate the heart, alcohol, coffee, tea, etc., and as far as possible from depressing emotions.

The first evidence of degenerative change may be counteracted by developing the muscular fibres under both fresh air and exercise. A systematic course of stimulation by digitalis, strophanthus, or other heart tonic, will often bridge a patient over a collapse and secure a new lease of life.

Aortic Stenosis.

(Angustia Aortæ.)

Obstruction at the aortic orifice occurs most frequently in consequence of arteriosclerotic or atheromatous change. Aortic stenosis may be congenital, and the conditions which develop in after-life are often based upon congenital substrata, particularly upon arrests of development or upon acquired anomalies in connection with tuberculosis, hæmophilia, myocarditis, and fatty degeneration. There is often hypoplasia of the heart.

The obstacle is offered usually by thickening or calcification of the aortic valves. The process commonly begins in the aorta and travels down in the course of time to involve the valves, which lose their pliability and become converted into hard membranes of almost cartilaginous feel. Not infrequently a calcareous mass may be felt or may be seen as an opaque mass upon the surface of the finger inserted into the pocket or sinus behind the valve. Sometimes the whole valve has the feel of a rigid unyielding mass, and the finger nail or knife drawn over the surface elicits the grating sound of contact with mineral matter.

In the more exceptional cases the tissues of the valves suffer change under the processes of endocarditis. In this process the valve tissue is thickened so that the structure becomes opaque. The edges are shrunken or rolled over upon themselves, or thrombotic matter is deposited upon the surface of the valves to constitute verrucose or cauliflower excrescences. Sometimes two segments may become agglutinated so that the play of the valves is prevented. Endocarditis does not usually begin in the aortic valve, though exceptional cases of this localization have been reported. As a rule, the process extends from the mitral valve, so that the affection of the aortic valve represents a subsequent attack of rheumatism, or other infection, or a later period in the history of heart disease.

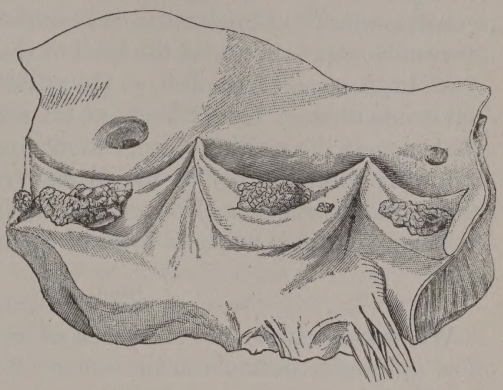

Fig. 12.—Verrucose Endocarditis of the Aortic Valves. The figure shows also the site of the orifices of the coronary arteries.

The size of the opening may be itself reduced in varying degree, so that the orifice, which usually admits the point of the finger, may scarcely permit the passage of a lead pencil, quill, or sound; or the opening which is left may be more or less circular, triangular, irregular, or show a small narrow slit like a buttonhole. Sometimes the valves unite at the edges and constitute a more or less rigid diaphragm and, with the opening in or near the centre, form a kind of funnel which protrudes into the aorta.

The "funnel" form and the slit or "buttonhole" orifice are changes in shape most advantageous to the discharge of the greatest possible amount of fluid according to the principles of mechanics. So long as the valves are pliable, the funnel is formed with the discharge always in the direction of the blood current. As the valves become rigid the slit is formed and this (the buttonhole) is always more favorable to the passage of fluid through an obliquely stretched septum than is a round opening (Campbell).

Aortic stenosis implies affection of the valve more strictly than aortic insufficiency, for insufficiency may occur independent of any affection of the valves from dilatation of the aorta, whereas stenosis almost of necessity implies affection of the valves themselves.

As the process most frequently depends upon atheromatous

change, aortic stenosis is more common in advanced life. Exceptional cases, however, are found to be due to cicatricial masses, from myocarditis, and still rarer cases are caused by compression from without. Ditthauer reported a case of compression of the aorta at its origin by an enlarged gland. Chevers and Vulpian reported cases of obstruction from thickening of tissue below the annular ring in the interventricular septum, at the level of the base of the mitral valve.

As in the case of stenosis at the mitral orifice, aortic stenosis seldom exists alone. Insufficiency of the aortic valves is frequently the sole lesion at the aortic orifice, but stenosis is nearly always associated with some degree of insufficiency, as the same lesion which causes the obstruction also permits regurgitation. A pure stenosis of the aortic orifice is a rare form of valve lesion.

Symptoms.

Whatever may be the character or cause of the obstruction, the effect is to offer obstacle to the escape of blood from the left ventricle. In this way extra work is thrown upon the left ventricle, which immediately suffers dilatation and soon undergoes hypertrophy. This hypertrophy may be very considerable, but it never assumes the magnitude of aortic insufficiency, if only because of the fact that the heart muscle itself is so imperfectly nourished. The obstacle which is offered to the escape of blood interferes with the blood supply of the heart through the coronary arteries. Moreover, the lesion itself often depends directly upon arteriosclerosis, which, by diminishing the resilience of the aorta, also interferes with the feeding of the coronary arteries.

As already stated elsewhere, a considerable degree of hypertrophy may exist and fail to make itself manifest by much increase in the appreciable outlines of the heart. Thus, while the dulness in aortic stenosis is somewhat increased to the left, the apex of the heart is seldom dislocated to any great extent. It beats in the natural position or deviates but slightly from the mammillary line. Nevertheless the stroke is found also usually in the sixth intercostal space, and this downward dislocation indicates the predominant hypertrophy of the left over the right ventricle. But the hypertrophy may make itself manifest in the increased action of the heart. The stroke is stronger and the area of impact is greater. Potain finds in this strong impulse, with scarcely appreciable hypertrophy and with but slight displacement of the apex, a conjunction of circumstances which speaks in favor of aortic stenosis. The hypertrophy of the heart is rarely so extreme as to protrude the wall of the thorax and anything like a vaulting of the thorax, which could be

observed of course only in the young, would indicate a complication with aortic insufficiency.

The hypertrophy of the left ventricle sometimes, though rarely, perfectly compensates the lesion, usually but imperfectly, and in all cases when the compensation begins to flag, the left ventricle undergoes dilatation, often to such an extent as to induce relative insufficiency of the mitral valves, but in any case to such degree as to lead to dilatation. This dilatation is followed in turn by dilatation of the left auricle and interference with the circulation of the lungs. Hereupon now ensues dilatation and hypertrophy of the right ventricle, which also for a time assists the defective compensation. The hypertrophy of the right ventricle is rarely so extreme as to be appreciable by percussion, though the dilatation which supervenes later may make itself manifest, with the development of insufficiency of the tricuspid valves, by the signs of heart failure.

The discharge of blood under the powerful action of the hypertrophied left ventricle through the contracted orifice, gives rise to a fremitus or thrill which may be distinctly perceived by palpation. This *frémitus cataire* is felt most distinctly in the region of the aorta at the second interspace to the right of the sternum. It corresponds in time to the systole of the ventricle, is therefore synchronous with the first sound, and, appreciated in this region and at this time, constitutes one of the most valuable signs of aortic stenosis.

The forcing of the blood through the contracted orifice also develops a murmur which is heard in greatest intensity at the second interspace to the right of the sternum and synchronous, of course, with the systole or first sound of the heart. The murmur is usually long drawn in correspondence with the slow discharge of blood through the contracted orifice, but is not usually propagated into the carotid arteries. The tone of the murmur, whether soft or rough, depends upon the character of the lesion. Sometimes it is particularly strident, more rarely rasping or sawing, sometimes it is whistling or hissing; sometimes it strikes a musical note; sometimes, again, it is so faint as to be perceived with difficulty, sometimes so intense as to disturb the repose of the patient or as to be audible at some distance from the body. It is heard all over the heart and is often so loud as to drown the sound of the closure of the mitral valves. The duration of the murmur depends to a considerable extent upon the condition of the heart muscle.

In the absence of any insufficiency the second sound is feeble. Sometimes it is scarcely audible, as the lesion which produces the obstacle retards, though it may not prevent, the closure of the valves.

v. Noorden made an accurate estimate by means of the acoustic

method of Martius of the relation in time of a heart murmur in a case of aortic stenosis. The systolic murmur began distinctly later than the first heart sound, and this point can be made of value in the diagnosis of aortic stenosis.

A characteristic condition is observed in the pulse. The issue of blood under the forcible contraction of the ventricle through the narrow orifice is long drawn. The pulse, therefore, is retarded and is usually small and sometimes so hard as to be unobliterable. Such a condition is sometimes said to constitute the wiry pulse. But it is the slowness rather than the hardness which is obtrusive. These peculiarities are most manifest in the sphygmograph which shows an oblique line of ascension and broad apex which forms a kind of rounded plateau and which represents the propulsion of blood through the contracted orifice, and a descending line without much interruption by dicrotism on account of the strong arterial tension and the slow diastole of the vessel. Lüderitz showed that contraction

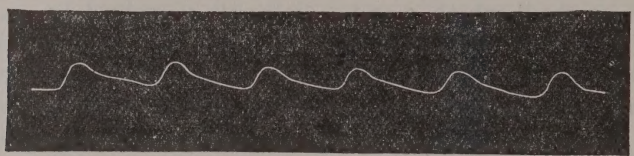

FIG. 13.—Pulse Curve in Aortic Stenosis. Short ascent, rounded apex, great resistance; wiry pulse.

of the aorta, when not extreme, does not lower the average blood pressure in the aorta, but that the heart by reason of its reserve force may fully overcome the obstacle. In this process the systole is lengthened and with it the corresponding anacrotic part of the pulse curve, while the apex curve is more rounded. The heart rhythm remains often unchanged. Sometimes it is lengthened, sometimes there is bigeminism and trigeminism. Retardation of the pulse wave does not occur, but there is a dislocation of the apex of the trace because the ascent is slower.

Steel distinguishes two varieties of pulse in aortic stenosis, the anacrotic and the "*pulsus bisferiens.*" This latter variety resembles the pulsus celer of aortic insufficiency. Both the ascent and descent are sudden and straight, and the recoil shows an acute angle. In the two cases marked by the pulsus bisferiens there was besides the stenosis pronounced insufficiency. But this pulse has also been observed in cases of pure stenosis.

The radial pulse in the case of insufficiency showed extremely characteristic peculiarities. It was the *pulsus altus et celer.* Nothing of this is seen in the case of stenosis. The pulse is small and

slow. The pulse peculiar to aortic stenosis is the *pulsus tardus*, because of the slow distention of the artery under the contraction at the aortic orifice and because of the rigidity of the artery, which is usually found in association with stenosis of the orifice. When the apex stroke is strong it stands in striking contrast with the relative smallness and tardiness of the pulse. Moreover, the frequency of the pulse is diminished because the stenosis at the orifice of the aorta interferes with the blood supply of the coronary arteries and so reduces the frequency of the action of the heart (Leube).

The general symptoms which distinguish aortic stenosis belong especially to arteriosclerosis. Patients suffer with frequent attacks of palpitation which are often paroxysmal and persistent. Dyspnœa may occur upon the slightest effort or without effort. Pain in the region of the heart is frequent and is often severe. In fact, attacks of stenocardia are not uncommon. These attacks, at first comparatively slight and recurring at long intervals, become more frequent and severe in the later course of the disease. The general process is indicated further by the pallor of the face, anæmia, and exhaustion after slight efforts.

Curschmann calls attention to the development of a cicatricial paranephritis in disease of the aortic valves, especially in chronic endocarditis of the semilunar valves and in certain atheromatous changes of the aorta. These cases are marked by severe neuralgia and frequently agonizing attacks of pain in the domain of the lumbar nerves, especially of the ileo-hypogastric nerve.

These attacks usually follow renal hæmaturia. They result from the paranephritic changes which develop in consequence of renal emboli. The cicatrizing tissue becomes thicker after every attack and finally makes pressure upon the subjacent ileo-hypogastric nerve. The pathogeny is confirmed at the autopsy. The only point in differential diagnosis concerns certain forms of nephrolithiasis.

Diagnosis.

The diagnosis, which may be indicated by the evidence of arteriosclerosis, is determined chiefly by the physical signs, to wit: slight increase in dulness, chiefly to the left, dislocation of the apex downwards rather than to the left, distinct increase in the action of the heart, palpable thrill and systolic bruit at the base of the heart, heard in greatest intensity at the second right interspace close to the sternum and synchronous with the first sound of the heart. The diagnosis of a pure stenosis is easy. But such cases are rare. The association with insufficiency, which is the rule, modifies the symptoms. The characteristic pulse disappears so that the pulsus tardus

is substituted by the pulsus altus et celer of a predominant insufficiency. In such cases the diagnosis of the stenosis may be impossible, as the systolic murmur may be derived from a secondary dilatation of the ascending aorta.

Congenital stenosis is distinguished by the permanency of the foramen ovale and the ductus Botalli, which leads to stasis in the right heart, hypertrophy of the right heart, and cyanosis. As most individuals thus affected succumb during the first days of life, the question of differential diagnosis scarcely comes into question (Leube).

A contraction of the aorta, congenital or acquired, may present the same symptoms. Aufrecht reports the case of a laborer, aged forty-four, in whom there had been established the diagnosis of stenosis and insufficiency of the aortic valves, which at the autopsy were found to be perfectly normal. The regurgitation was due to the fact that the blood was not able to insinuate itself behind the valves and close them for the reason that the valves were overlapped by a circular mass of sclerotic tissue in the aorta. A somewhat similar case was reported by Kretz where a contraction of the aorta gave rise to the symptoms of a combined heart lesion.

An extracardial systolic souffle, that is, the so-called accidental murmur of anæmia, is usually more soft and superficial and is unattended with fremitus or hypertrophy. Besides, the bruit of anæmia does not show the peculiar pulse, pulsus tardus, of obstruction in the aorta.

Aneurysm of the aorta shows usually more decided increase of dulness, especially to the right of the sternum at the base of the heart, while the pulsation can often be felt in the jugulum when the finger is pressed behind the notch of the sternum. Aneurysm is sometimes attended with inequalities of the pulse on the two sides. In aortic stenosis the pulse is small and hard, or if not hard is always slow. Paroxysmal attacks of palpitation, dyspnœa, stenocardia, vertigo, or syncope, which occur more frequently in stenosis, facilitate the diagnosis.

Prognosis.

Where the stenosis is slight and compensation is readily established, the prognosis is not unfavorable, and aortic stenosis, even with a light degree of insufficiency, may exist for some time without serious symptoms. In fact, a considerable degree of contraction at the aortic orifice may remain latent for a long period.

Sometimes the condition is discovered only accidentally during the search for a cause of attacks of vertigo or syncope in older people, more frequently in explanation of stenocardia in younger people.

Bamberger reports cases of sudden death with epileptiform convulsions, which could be explained by anæmia of the brain under the extreme angustia aortæ which was disclosed upon the post-mortem table.

Aside from the character or extent of the lesion and the general evidence of arteriosclerosis, the prognosis will be determined largely by the surroundings of the patient. Where the life may be regulated, the condition may last for many years. In fact, it may be said in a general way that the prognosis of aortic stenosis is better than that of aortic insufficiency. Of course, an extreme stenosis would furnish a graver prognosis than a slight insufficiency. Practically, as stated, the conditions coincide and the prognosis is determined by the preponderance of one or other factor, by the condition of the heart muscle, and by the extent of the arteriosclerosis elsewhere.

Treatment.

The treatment must have special reference to the conservation of the muscular tissue, especially by sparing the heart unnecessary effort or emotional disturbance. This conservation is best secured by rest. It must be remembered, at the same time, that excessive rest favors degenerative, especially fatty and arteriosclerotic change. Patients fortunate enough to control their surroundings soon learn to regulate the rest and exercise according to the condition of the heart. The subject of aortic stenosis should be especially protected against disease of the lungs; that is, in younger life, against exposure to measles; and at any time against exposure to tuberculosis.

In the treatment of the stenosis tumultuous action may be restrained by the application of cold or by the occasional administration of sodium bromide.

In relief of the underlying arteriosclerosis, which is the most common cause of the condition, the iodides should be administered regularly, preferably sodium iodide in doses of grs. v.–x. in a wineglassful of milk before meals. Attacks of stenocardia may call for the use of amyl nitrite, which may be inhaled from a pocket handkerchief, or of nitroglycerin internally in doses of one or two drops of the one-per-cent. solution three or four times a day. Dyspnœa may be relieved by the diffusible stimulants, brandy, ether, camphor, valerian, etc. Anæmia is best combated by the administration of iron in the form of the tincture of the chloride, fifteen to thirty drops in a glass of sweetened water three times a day, or the saccharated carbonate in the dose of a teaspoonful three times a day. When the compensation begins to give way the action of the heart may be sustained a long time by the timely use of strychnine, caffeine, strophanthus, or digitalis.

Lesions of the Valves of the Right Heart.

Tricuspid Insufficiency.

Lesions of the tricuspid valves were of later recognition than those of the valves of the opposite auriculo-ventricular orifice, chiefly because they are so much more rare. In fact, anatomical lesions of the tricuspid valves are found only once or twice in one hundred cases of valve disease. But the lesions are due to the same cause, to wit, to endocarditis in the course of some infection or to atheroma. The tricuspid valve is rarely the sole seat of disease. Blix, however, has reported a case of septic endocarditis affecting this valve. The affection of the tricuspid is usually consecutive to affection of other valves, especially of the mitral valve. It is when the process is general and widely disseminated through the heart that the tricuspid is also involved. The lesions may become so extensive in other valves as to take life and nevertheless entirely spare the tricuspid valve.

But, as stated elsewhere, this exemption of the tricuspid valve applies only to extra-uterine life. In fœtal life the tricuspid valves are most frequently affected. This preference to attack is ascribed to the fact that the tricuspid valves have in fœtal life the most work to do. The blood from the umbilical vein evades the left heart as much as possible and leaves the mitral valves comparatively inactive. So most of the cases of tricuspid-valve affection due to endocarditis date from intra-uterine life, and are thus congenital.

Moreover, the processes of atheroma rarely attack the tricuspid valve. The aortic valves may be transformed into rigid cups or cusps, the mitral valves may be changed into calcareous plates, while the tricuspid valves still retain their pliability and resilience.

Nevertheless, with all these exemptions the tricuspid orifice is frequently the seat of disease. In this process the valves themselves may not be directly affected. In fact, their structure usually remains intact, but the ring of insertion, more especially the muscular tissue which regulates it, becomes so affected as to render the valve incompetent to close the orifice. It was Jendrin who first observed and distinguished by the term it still bears, the relative insufficiency of the tricuspid valves. This acute observer noticed the change especially in connection with dilatation of the right ventricle, in which process it was seen that the ring of insertion at the base of the valves was so much widened as to prevent perfect apposition under the pressure of the blood. Dilatation of the right ventricle occurs as the result of many different processes, but ensues most frequently as a

direct consequence of affection of the mitral valve. In either obstructive or regurgitant lesion at the mitral valve the blood accumulates in the left auricle, in the pulmonary veins and lungs, and in the pulmonary artery. Under this pressure the right ventricle suffers dilatation and undergoes hypertrophy in order to overcome the pressure in the pulmonary artery. The dilatation at first is not great and the lesion may be compensated by the reserve force of the right ventricle. With the exhaustion of this reserve force in the course of time, the ventricle undergoes hypertrophy and thus compensates the lesion. It is when this hypertrophy begins to give way under degenerative change that the cavity of the right ventricle yields, and finally to such an extent as to make the tricuspid valves incompetent.

This process may be materially hastened by the changes of myocarditis. The fact is, all valve lesions are accompanied by changes in the myocardium generally of progressive character. The degenerative change is usually insidious and slow; there may be long periods of quiescence, but the tendency is toward advance and extension of the disease process. Certain infections, notoriously diphtheria, typhoid fever, scarlet fever, precipitate the degenerative changes of myocarditis and thus induce in an earlier stage the relative insufficiency of the tricuspid valves. Other causes are very rare. Gräffner reported a curious case in which a syphilitic gumma was transformed into a tendinous diaphragm which traversed the ventricle and involved the anterior segment of the tricuspid valve, whereby the valve was fixed and rendered incompetent.

Insufficiency of the tricuspid valves permits the blood to regurgitate into the right auricle. With its feeble muscular force the right auricle soon yields to the pressure and under the contraction of the right ventricle the blood wave is propelled back through the auricle into the venæ cavæ, and finally into the veins of the neck and body.

Morbid Anatomy.

The changes of endocarditis when encountered are the same as those in the case of the mitral valve. The structure of the valve itself is thickened, opaque, rolled over upon its borders and reduced in size. Sometimes, more rarely, the surface is covered with the same cauliflower excrescences and verrucosities. In other cases the disease process expends its force mainly upon the columnæ carneæ or tendinous cords which it reduces and fixes so as to bind down the valve and make it more or less immobile. In rarer cases the tissue of the valve may be perforated, sometimes under powerful muscular effort —the perforation in these cases implying a previous weakening of the

structure—or again there may be rupture of an aneurysm or abscess, or disintegration of a thrombotic deposit by molecular necrosis.

But in the great majority of cases no direct lesion of the valve is encountered. It is seen, however, that the size of the orifice is too big for the valves. Instead of two, three or four fingers may be pushed through the right ariculo-ventricular orifice. Accurate measurement of the valves and orifice may show a distinct disproportion. No exact estimate may be made, however, of the size of the orifice by such crude methods, or by any method which may be used post mortem, as at this time the contractile muscular tone, which asserts itself in life to adjust the valves and compensate the lesion as much as possible, is now entirely lost.

Symptoms.

The dilatation and hypertrophy are shown in the increase in the diameters of the heart, especially to the right, and are evidenced also in the more distinct pulsation in the right ventricle at the ensiform cartilage, or as it may be conducted over the epigastric region. The hypertrophy may never become extreme; it yields much sooner than in the case of the left ventricle to show dilatation in the signs of beginning heart failure.

The regurgitation of blood through the right auriculo-ventricular orifice under the contraction of the right ventricle, produces a murmur which is heard in the greatest intensity in the region of the ensiform cartilage, the nearest surface point of the tricuspid valves, and in the direction of the blood current in the heart partially dislocated by hypertrophy or dilatation. The murmur is heard in greatest intensity at the lower half of the sternum and distinctly to the right of it. When in an exceptional case it is heard to the left of the sternum it may give rise to confusion with mitral insufficiency. It is seen, however, that the murmur in the case of tricuspid insufficiency persists to the right of the sternum and may be followed as far as the right axillary line, which is never the case with insufficiency of the mitral valve. The murmur is usually soft and blowing in character and may be so faint as to be heard with difficulty. In fact, it is sometimes entirely absent. It is only in exceptional cases that the murmur is rough and rasping.

In consequence of the fact that a smaller amount of blood is propelled into the pulmonary artery, the second valve sound is weak. When a pulmonary valve sound has been previously accentuated in consequence of lesion of the mitral valves, the reduction of this compensation and comparative faintness of the pulmonary valve note may indicate the beginning of tricuspid insufficiency.

The Venous Pulse.—Reference has already been made to the venous pulse which is sometimes so distinctive of tricuspid regurgitation. The superior vena cava and the innominate veins have no valves. The refluent blood wave is therefore propelled into the venæ cavæ, sometimes only into the superior vena cava, always in greater quantity into the superior than the inferior because the superior lies more directly in the line of propulsion and because the inferior vena cava is partly protected by the Eustachian valve. The true venous pulse begins in the jugular and subclavian vein. The jugular vein is furnished with a large valve at the sterno-clavicular junction. This valve long resists the pressure of the refluent blood. In the course of time, however, the vein swells below this point to show a tumor of the size of a walnut, soft and pulsating, bluish in color, the so-called bulb of the jugular vein, at the root of the neck in the supra-clavicular fossa, just outside the sterno-cleido-mastoid muscle. When the valve of the bulb has been overcome, the pulsation extends further into the course of the jugular vein, which now throbs visibly throughout its whole course in the neck. This pulsation also extends into the other veins, especially into the external jugular and subclavian, sometimes as far as the facial and axillary veins. The venous pulse is most pronounced in the internal jugular vein and is more distinct upon the right side than the left, because the right innominate vein is nearly a direct continuation of the vena cava, while the left makes an angle with it.

Distinct pulsation of the cervical veins is almost pathognomonic of tricuspid insufficiency. A feeble pulsation is not so distinctive, as it may represent only the systole of the auricle. The jugular may be distinguished from the carotid pulse by the fact that it may be obliterated so much more easily by pressure and that all pulsation necessarily ceases in the vein above the point of pressure. It would be difficult to make such pressure upon the carotid artery as entirely to obliterate the pulsation.

Regarding the venous pulse, Riegel finds from his examination of a large number of individuals, sick and sound, that rhythmic movements of the veins in the neck by no means indicate only insufficiency of the tricuspid valves—as such rhythmical motions are not infrequently seen in cases in which there is no change whatever of the tricuspid valves and in fact in which there is no trace of any heart disease at all.

These motions depend upon the afflux of venous blood to the heart. Thus with the beginning of the diastole of the auricle the afflux of venous blood is easiest and therefore in this period the veins collapse; toward the end of the systole the afflux becomes more difficult, there-

fore the veins begin gradually to swell and this swelling is increased at the end of the diastole with the beginning of the systole of the ventricle.

It is seen thus that the normal venous pulse does not depend upon an insufficiency of the valves of the veins of the neck nor upon a refluent wave, but is simply the expression of the emptying of the venous blood into the heart, which is now helped and now hindered by the individual phases of the heart action. The true venous pulse in the narrowest sense is that motion in the veins in the neck which is produced by a centrifugal blood wave driven back into the jugular vein from the right ventricle. This pulse Bamberger considered as characteristic of tricuspid insufficiency. But the venous pulse does not of itself prove a tricuspid insufficiency, as the jugular vein, even in normal condition, shows pulsating motions.

But there is a characteristic pulse for tricuspid insufficiency, a pulse which alone demonstrates with certainty incompetence of the tricuspid valves. The characteristic feature of this pulsation does not lie in the form, nor does it lie in the size and strength of the venous pulse. It lies alone in its temporal relations. The true venous pulse of tricuspid insufficiency distinguishes itself by the fact that it is presystolic-systolic. It is presystolic, because the auricle cannot fully empty itself into the distended ventricle, and systolic as a refluent wave from the ventricle. Thus it occurs both with the systole of the auricle and with that of the ventricle, and it is only with the diastole of the ventricle that the afflux of venous blood is helped and there ensues a diastolic venous collapse.

This typical conduct of the venous pulse in tricuspid insufficiency occurs only in the presence of good power in the heart. It is only in these cases that the sphygmograph shows in the jugular vein, not only a presystolic but also a positive systolic wave (Riegel).

When the slighter resistance of the Eustachian valve has been overcome, the blood is propelled into the inferior vena cava and the venous pulse may be recognized in the liver and in the femoral veins.

This palpation of the liver furnishes valuable evidence in diagnosis, and all the more valuable because the venous pulse in the liver may be appreciated at an early period of tricuspid insufficiency. The fact that the veins in the liver are unprovided with valves explains the occurrence of pulsation in the liver before it is perceived in other veins. When both hands are applied over the region of the liver the whole organ may be felt to beat under the systole of the heart.

The pulsation of the liver in tricuspid insufficiency was noticed by Sénac, Kreysig, and especially by Friedreich, who showed that it was caused by an expansion of the liver itself. The maximum pulsa-

tion is felt at the level of the left lobe, not because of approximation to the heart—that is, not because the movement is communicated—but because of the easier expansion of the lesser lobe. The pulsation is like that of an angioma or erectile tumor. When the liver is seized between the two hands, one below and one above, it is seen that it enlarges itself regularly with each ventricular systole. In this way the distention due to tricuspid insufficiency is distinguished from the pulsations propagated from the abdominal aorta.

Venous pulsation requires with tricuspid insufficiency a strong action of the left ventricle. The reason why pulsation of the liver is observed so rarely is because these conditions seldom coincide.

The pain in the region of the liver is the result of tension of the capsule from swelling of the liver. The spleen swells in the same way, and in protracted cases the interference with nutrition leads to the same hyperplasia of the connective tissue with subsequent contraction and cyanotic induration.

Cardiac cirrhosis is nearly always attended by ascites, and when ascites is the solitary expression of dropsy it gives rise to great embarrassment in diagnosis.

The paucity of the valves in the veins makes much more manifest the force of respiration. The venous pulse is everywhere distinctly increased during inspiration and diminished during expiration. The patient, therefore, may make an irregular venous pulse manifest by drawing a long breath.

The sudden closure of the valves and the sudden tension of the walls of the veins give rise to a definite sound, which is most distinctly appreciated at the bulb of the jugular vein. Bamberger first called attention to this sound of the veins and showed that at a certain stage when the auricle is sufficiently powerful, the systolic sound may be preceded by a shorter presystolic note. These periods of time may be recognized by comparison with the pulsating carotids or in later periods of irregularity in arhythmia may be appreciated only by some suitable apparatus (Kovacs). The pulsation in the crural vein may be seen to precede slightly the pulsation of the crural artery, as the distance in the blood column in the adult is 20 cm. less.

Under the powerful action of the right ventricle, the vibration of the walls of the crural vein produces a distinct sound which may also, under a strong auricle, be preceded by a slight presystolic note, the crural vein double sound. Insufficiency of the valves of the veins is marked also by a double murmur, and quite complex acoustic phenomena may be produced by a combination of these sounds with the simultaneous sounds in the crural arteries already described in connection with aortic insufficiency.

The decrease in the force of the right ventricle soon leads to lowering of pressure in the whole venous system and stasis of blood in the systemic veins. The signs of stasis show themselves in ecstasia of the veins and in defective aëration of the blood, in duskiness of the whole surface with the development often of pigment matter, which accumulates especially in the internal organs and leads in highly vascular structures, in the lungs, liver, etc., to the well-known cyanotic induration.

Under the defective nutrition which now supervenes, serum escapes from the blood-vessels, at first at points most distant from the circulation, as about the ankles; but under the progressive course of the degenerative change in the heart the fluid accumulates in the lower extremities, mounts the trunk, distends the genital organs, invades the serous sacs, escapes into the subcutaneous tissue everywhere, constituting a general anasarca.

Cardiac Asthma.—The defective supply of oxygen to the tissues leads to dyspnœa, which is shown first in short breath and panting respiration after effort, and which later occurs spontaneously and assumes finally such proportion as to render the individual incapable of all effort and to disturb his rest at night. In bad cases the patient is no longer able to lie down; in the worst cases he may no longer lie in bed, but must sit up in a chair near an open window with the chest bare to every breeze. Sometimes in this condition the patient can sleep only when he is fanned constantly throughout the night.

In other cases the dyspnœa is less continuous; it occurs in paroxysms which arouse the patient at night shortly after first falling asleep, under the apprehension of suffocation. These paroxysmal attacks constitute the so-called cardiac asthma, which is entirely independent of spasm of the bronchial muscles and is due wholly to heart failure. In these attacks the patient suddenly starts in his sleep, must immediately rise in bed and begin the painful struggle of inflating the lungs. In this struggle the skin is covered with a clammy sweat, the eyes are suffused, the veins throb in the neck, the expression is wild and anxious; sometimes the face wears the look of desperation. The breathing is so rapid and superficial as to prevent the use of the voice, and any attempt that is made is expressed in broken syllables. Effort of any kind aggravates the difficulty of breathing.

The action of the heart is usually tumultuous at first, but becomes later, with the subsidence of the attack, more and more weak. In patients still provided with any amount of fat, the action of the heart is faint or absolutely imperceptible. There is often delirium cordis. The pulse is correspondingly feeble and weak, shows distinct arhythmia, vibrates like a loose thread, and is easily obliterated by the

slightest pressure. It may fade away entirely when the arm is held at right angles from the body. The whole surface is clammy, the extremities are cold and blue. The patient may succumb in any of these attacks from heart failure, which is usually immediately preceded by œdema of the lungs. Usually, however, the attack gradually subsides to leave the patient in the same condition as before. Such attacks may occur every night, or every few nights, or may later set in at any time during the day.

But œdema of the lungs is a frequent complication of tricuspid insufficiency independent of attacks of cardiac asthma. Most of the cases of tricuspid insufficiency are found in connection with the lesion of the mitral valve. Welch developed œdema of the lungs in some experiments in which he weakened the action of the left ventricle, leaving the right intact. Interference with the escape of blood from the left ventricle leads to the same result. The extreme stasis which occurs in tricuspid insufficiency develops œdema of the lungs in the course of the general anasarca. The occurrence of this œdema is announced at all times by interference with respiration, which becomes superficial and shallow, and this interference renders necessary the appeal to all the auxiliary muscles, with strained efforts at respiration, a sense of suffocation, anxiety, duskiness of the surface, and cyanosis. During the existence of the œdema the sputum is thin, foamy, and tinged with blood. It contains also, as points of diagnostic value, œdematously swollen cells of desquamated epithelium, often studded with particles of coal dust. These bodies are sometimes distinguished as the cells of heart failure.

Inspection in these cases shows the limited excursion of the chest. Percussion gives dulness below, increased resonance, even tympanites, above the effusion. Auscultation discloses universal mucous and submucuous râles, which drown all other sounds, which may be felt by palpation, and may be heard by the patient himself and often by others in the vicinity.

The *diagnosis* is sometimes apparent in the systolic venous pulse in the neck or in the bulb of the jugular vein which protrudes as a pulsating tumor, sometimes, after violent effort, such as coughing, as big as the end of an egg. The true venous pulse is systolic and is much increased below the seat of compression when the veins are compressed in the neck. The false venous pulse is due to the contraction of the hypertrophied auricle. This pulsation is presystolic and is seen sometimes in mitral obstruction. The false venous pulse, instead of being strengthened, is weakened by compression, as in this way less blood is allowed to enter the auricle.

The condition is also pretty clearly indicated in the presence of

the signs of extreme heart failure, dyspnœa, cardiac asthma, cyanosis and dropsy, occurring in the later course of some other valve lesion, of pericarditis, myocarditis, arteriosclerosis, Bright's disease, etc.

The physical signs are decided enlargement from dilatation, with arhythmia and tachycardia, signs which are manifest also in the pulse, visible pulsation at the ensiform cartilage, with a bruit, usually of soft or blowing character, heard in the greatest intensity under or at the right of the ensiform cartilage and synchronous with the first sound of the heart.

Plain as the diagnosis may be in an advanced case, in the beginning it may be very difficult. The practitioner who makes a diagnosis of primary or pure tricuspid insufficiency will nearly always be disappointed at the autopsy, as the lesions of endocarditis may be practically excluded from consideration. Valve lesions of this character are sometimes found in the fœtus and in the dead-born child, but are encountered only in the most exceptional case in the adult and then only after previous affection of other valves. The clinician will also be slow to make a diagnosis of relative insufficiency unless the condition be justified by valve lesion of the left heart or by myocarditis (Bright's disease).

Sturges remarks upon the presence of a murmur at the ensiform cartilage from functional insufficiency of the tricuspid valve in consequence of stasis in the lungs as the first sign of endocarditis affecting the mitral valve in childhood. The murmur peculiar to the mitral valve develops itself later in the course of the affection.

The *prognosis* is always bad for the reason that the hypertrophy of the right ventricle has hitherto been able to compensate the original lesion. Insufficiency of the tricuspid valves, which is, as stated, nearly always relative, indicates dilatation, which is due in turn for the most part to degeneration of the muscular tissue. The outlook will now depend upon the immediate surroundings of the patient and especially upon the response of the heart to digitalis. The prognosis is better, therefore, in the case of patients who fall into the hands of a physician who may put the patient upon digitalis for the first time. When the heart no longer responds to digitalis, the end is not far off. In a general way it may be said that tricuspid insufficiency is the last link of a long chain of disease processes in the heart. The breaking of the compensation in the right ventricle is literally the rupture of the last link in this conservative process.

Nevertheless, under proper surroundings and with the proper use of heart stimulants, especially digitalis, the patient may readily improve from the most dangerous stage of heart failure. He improves for a time, and even for a long time.

Treatment.—The treatment demands absolute rest that the mechanism of the heart's action may at least adjust itself as much as possible to the altered conditions of the circulation. The patient may be allowed, however, under all circumstances to take what position will give him the most comfort. These are the cases which are especially benefited by the use of the heart stimulants, and an immediate collapse, indicated by an attack of cardiac asthma or œdema of the lungs, may be bridged over by the use of caffeine subcutaneously, especially the sodium benzoate in the dose of grs. iij. to v. Hereupon resort is had at once to digitalis in the form of powder, infusion or tincture, and the dose is graded according to the condition of collapse. It is astonishing how rapidly relief is offered in this way. Thus the cyanosis may entirely disappear, the dropsy may fade away, and the difficulty of breathing become so little marked as to be present only after unusual effort; so that the patient may now lie down in bed and secure the restoration of a sound sleep. The management of this condition in detail will be further described under the general treatment of valve lesions.

Tricuspid Stenosis.

Stenosis at the right auriculo-ventricular orifice is one of the rarest of the heart lesions. Most of the cases which have been seen belong to fœtal life, and though occasional cases have been reported, it is doubtful if a purely stenotic lesion has even been produced by endocarditis after birth. In nearly all the cases the condition has been found associated with tricuspid insufficiency, and in nearly all also with lesions of the left side of the heart. Duroziez reports the isolated case in medical literature of uncomplicated tricuspid stenosis.

As the condition belongs rather to fœtal life, it is not surprising to learn that tricuspid stenosis is found in connection with other congenital lesions. Shipmann saw it in connection with other malformations of development, especially with perforation of the ventricles. Leudet found tricuspid stenosis 86 times in 100 cases in connection with or as a sequel to lesions of the left heart, always of the mitral valve. But Fenwick found it also in connection with lesions of the aortic valve. It is a curious fact that most of the cases reported have been found in the female sex. Fenwick found in the literature 46 cases, of which 41 were women. In 100 of the 117 cases in which the sex was mentioned in the collection of Leudet, 80 were women.

Etiology.—As intimated elsewhere, fœtal endocarditis is assumed to be an expression of fœtal rheumatism, and if the previous view of

the nature of rheumatism be correct—to wit, that the disease is due to attenuated pyogenic micro-organisms which invade the blood from some manifest or cryptogenetic centre—it is possible to understand the existence of both rheumatism and endocarditis in fœtal life.

In the rarer cases in which the lesion has been acquired, it has been ascribed to the same cause as in the case of other valves. Thus rheumatism was said to be the cause of one-half of the cases collected by Fenwick, and of one-third of the cases cited by Leudet. Lyonnet found it in connection with chorea. Potain declared that he was able to find no explanation in half of all the cases (Petit). Leudet reported several remarkable cases in which the obstruction was caused by demands upon the tricuspid valves.

The obstacle offered to the escape of blood from the right auricle leads to accumulation, and to distention and dilatation of this structure. The right auricle is, however, but feebly prepared to compensate such a lesion. It undergoes ready dilatation and expands often to an extreme degree. This dilatation leads to rapid accumulation in the great veins and universal stasis in the body.

Morbid Anatomy.—The valves may show the same lesions as upon the left side of the heart, though it is questionable if any thrombotic deposits or excrescences may ever produce a pure stenosis. In nearly all the cases reported the obstruction was caused by adhesion or agglutination of segments which made of the valve a funnel protruding into the cavity of the right ventricle. The orifice of the funnel is of various size. Ordinarily the right auriculo-ventricular orifice will admit the passage of two or three fingers. In stenosis the orifice may be so contracted as to permit the passage of but one finger, or perhaps the insertion of but the end of the little finger. This condition represents about the extreme degree of contraction compatible with life. The right auricle is found dilated in extreme degree, with attenuated walls, while the right ventricle is comparatively small. The muscular tissue of the heart shows every stage of degeneration under the myocarditic processes associated with more or less extensive valve lesions in the heart. Congenital anomalies of various kinds, perforation of the septum, persistence of the ductus arteriosus, etc., may be encountered.

Symptoms.—As tricuspid stenosis almost never exists alone but is found in connection with insufficiency and with quite extensive lesions of other valves in the left side of the heart, the symptomatology is usually masked by the signs of affection of the other valves as well as by the evidence of degeneration in the heart muscle itself.

The fact is, the symptomatology of tricuspid stenosis is a matter largely of artificial construction. Rosenbach declares that he was

unable to produce any distinct train of physical signs by experimental occlusion of the auriculo-ventricular orifice. This observer found that the introduction of small rubber balls which could be expanded into balloons in the orifice on the left side, or even the introduction of a thick sound, failed to produce any distinct murmur even in a sound heart, whereas in a condition of disease mitral stenosis is usually attended with distinct murmurs and often with distinct and palpable thrill. This fact is urged as an objection to the schematic representation of symptoms in connection with the right heart. In this representation a bruit should be heard in the greatest intensity under or to the right of the ensiform cartilage, or in the fifth or sixth intercostal space, to the right of the sternum, and should be diastolic or immediately presystolic, as in the case of the similar lesion of the mitral valves.

A pure stenosis develops no hypertrophy of the right ventricle, which is found rather in a state of atrophy. Increase of dulness may, however, be distinctly recognized in the neighborhood of the right auricle, which is distended in extreme degree.

The prominent general symptoms are those of extreme stasis of blood. Dyspnœa is more or less continuous, and is often extreme. Attacks of cardiac asthma are frequent. There is evidence of cyanotic induration of the liver, spleen, and kidneys, with a sub-icteric tint, which is sometimes increased to a distinct jaundice, with ascites as evidence of obstruction of the liver, with oliguria and albuminuria indicative of stasis in the kidney, hebetude, headache, mental confusion, and light delirium, the result of insufficient blood supply to the brain and of light uræmic poisoning. The surface is usually cold and more or less cyanotic.

The *prognosis* is unfavorable. Tricuspid stenosis is found in connection with congenital lesion or with extensive infection of other valves, which has already exhausted the heart.

The *treatment* is that of heart failure as described in connection with other lesions or detailed under the general treatment of valvular affections.

Pulmonary Insufficiency.

Insufficiency of the pulmonary valves is rare, though not so extremely rare as stenosis of the tricuspid valves. One of the first cases was recorded by Chevers (1842), and the condition has been since repeatedly described by Frerichs, Stokes, Litten, Grawitz, and Barié. Gerhardt (1892) collected the reports of twenty-nine autopsies from the literature.

Insufficiency of the pulmonary valves is the condition in which

the valve fails to close the orifice and blood regurgitates from the pulmonary artery into the right ventricle.

Etiology.—The condition is most frequently congenital and is found in association with stenosis of the pulmonary valves or with other congenital anomalies. Among the most frequent of these anomalies is a change in the number of valves. Sometimes but two valves are seen and they fail to close the orifice. Sometimes there are supernumerary valves and some of these valves fail. In other cases there has been noticed persistence of the foramen ovale or of the ductus arteriosus, perforation of the interventricular septum, etc. In nearly all cases the condition is found conjoined with lesions of other valves, and often with stenosis of the same valves. All the congenital cases are found in association with stenosis.

The acquired cases are due to endocarditis in consequence of rheumatism or other infection. Sometimes the valves are perfectly sound but the orifice is dilated so that the condition is that of relative insufficiency. The cause in these cases is usually an arteriosclerosis which produces dilatation of the pulmonary artery, and the cause of the arteriosclerosis is for the most part syphilis. In rare cases pulmonary insufficiency has been ascribed to trauma.

Barié found the condition at every age, with the maximum frequency between eighteen and thirty-four years.

Morbid Anatomy.—The lesions are of the same character as in the case of the aortic valves. The process of endocarditis thickens and stiffens the valves; segments become agglutinated to the wall of the artery, or adjoining valves may become adherent. The changes of atheroma are sometimes observed, and molecular necrosis may bring about the same destruction of tissue with the production of aneurysm and rupture or with detachment of valves from their base. Thus v. Wahl reported a case of nearly entire destruction by acute endocarditis, and Bernhardt observed distinct cases of rupture as the result of trauma from violent effort, assumed to be from severe cough. Such a rupture implies, of course, a pre-existent destruction or partial destruction of the tissue of the valve. Schwalbe reported two cases of chronic endocarditis and one case of syphilitic affection of the pulmonary valves. The first case was probably due to a congenital defect of the valves which made of them a *locus minoris resistentiæ* for the endocarditic process. In the first case, in a woman aged sixty-eight, the right pulmonary valve was markedly shrunken and thickened. The second case was that of a man, aged sixty-four, who died of senile gangrene. The left pulmonary valve was present only as a rudiment. Its left border was adherent to the pulmonary wall, the right was free but furnished only a very narrow passage. The right ven-

tricle was normal in both cases. The case of syphilitic affection was a stenosis of the conus pulmonalis with destruction of a pulmonary valve. Three yellow gummata, whose character was established by Virchow, of the size of a pea, were found in the region of one pulmonary valve, which was present only as a whitish cord. In the neighborhood of this cicatrix there were seven gummata from the size of a millet seed to that of a pea; further, a small gumma in the anterior pulmonary valve, and a large one in the wall of the vessel at the base of the left valve. Both valves were much fenestrated at the border. Chevers, Benedikt, and Bristowe reported cases of atheroma of the walls of the artery as the cause of the condition, and Barié found in more than half the cases contraction at the pulmonary orifice. Stokes and Gouroud described cases of relative insufficiency from simple dilatation.

Symptoms.—The general symptoms are those which mark an aggravated case of heart lesion. The condition cannot long remain latent but must soon reveal itself by defective nutrition, insufficient aëration of the blood, with dyspnœa and cyanosis.

As extra work is thrown upon the right ventricle by the regurgitation of blood from the pulmonary artery, the right ventricle undergoes hypertrophy and may thus suffice for a time to overcome the lesion. The right ventricle gets, however, no assistance in this work, and sooner or later begins to flag in its force. Cases of pulmonary insufficiency are therefore distinguished by the early occurrence of affections of the lungs, which are especially manifest in catarrhal symptoms and hemorrhage. Frerichs emphasized the frequency of tuberculosis in this lesion. The modern tendency is to ascribe this affection rather to the complicating pulmonary stenosis.

Physical Signs.—The hypertropy of the right ventricle makes itself manifest by dulness, which reaches to and beyond the right border of the sternum, also by dislocation of the apex somewhat to the left, and by distinct systolic pulsation in the epigastrium and in the second left intercostal space.

The regurgitation of blood is marked also by a bruit which is heard with greatest intensity at the base of the heart in the second left interspace, and which is synchronous with the diastole or second sound of the heart. The reflux of blood may make itself manifest by a distinct vibratory thrill, which is appreciated as a diastolic fremitus. The murmur, as a rule, substitutes the second sound entirely. It is usually so loud that it may be heard over the whole heart, even as far as the apex; and if the lesion be conjoined with a similar lesion of the aortic orifice, it is louder than the bruit of the aortic valves. A systolic bruit heard with greatest intensity in the same region

may depend upon a coexistent pulmonary stenosis, or may represent, as remarked elsewhere, the bruit of mitral insufficiency propagated into the left auricular appendix, which in certain cases winds about and comes to lie in front of the pulmonary artery.

The bruit of pulmonary insufficiency is, however, by no means always so intense. It may be very faint. It may vary also in quality. It has been often likened to escaping steam. It may strike a musical note.

The pulse at the wrist is perfectly regular. Any irregularity is due to affection of some other valve. The peculiar pulse of aortic insufficiency is, of course, never seen.

The *diagnosis* is based upon the fact that the condition is congenital and is found usually in connection with other anomalies of the heart, or that when acquired it occurs usually as a sequel to lesions of of other valves. Pulmonary insufficiency should show the increase of dulness to the right and below, which belongs to hypertrophy or dilatation of the right ventricle, and should disclose a bruit heard in greatest intensity at the base in the second interspace at the left border of the sternum, synchronous with the first sound of the heart. The diagnosis becomes easier when the ventricle stands low in the thorax and the reflux is attended by a distinct palpable thrill. The early occurrence of affection of the lungs and of the signs of defective aëration of the blood may speak for the character of the lesion.

In insufficiency of the pulmonary valves, Gerhardt refers in differential diagnosis to the presence of a palpable pulsation of the right ventricle between the ensiform cartilage and the right border of the ribs; further, to increase of the diastolic murmur during expiration. The murmur of inspiration in pulmonary valve failures is lower in tone on account of the lessened rapidity of the circulation. Moreover, there may be heard at the most distant possible point from the heart, thus laterally at the right shoulder blade, a low double tone, which may be appreciated also, though only in rare cases, in aortic insufficiency. At the same point there may be heard in pulmonary insufficiency an interrupted vesicular breathing during inspiration, the so-called lung capillary pulse. Gerhardt succeeded in certain cases in representing this pulse of the lung capillaries graphically, thus showing the variations of pressure in the broncho-tracheal column of air, whereby it was observed that the curves were very much more dicrotic than normal.

Pulmonary insufficiency is separated from aortic insufficiency by the absence of the hypertrophy of the left ventricle, which is so extreme in the case of lesion of the aortic valves, by the situation of the bruit to the left of the sternum in the case of the pulmonary

valve, to the right of the sternum in the case of the aortic valves, and especially by the fact that the bruit which results from insufficiency of the pulmonary valves is never propagated into the carotid arteries, as is the case in the lesion of the aortic valves. The anomalies of the pulse, the Corrigan, the capillary pulse, etc., are peculiar to the lesion of the aortic valves.

Aneurysm of the aorta is distinguished by its systolic bruit and irregular murmurs, which are most intense either higher, as under the notch of the sternum, or further to the right, one or two inches from the right border of the sternum. The various murmurs do not so exactly coincide with the phases in the action of the heart. Aneurysm usually shows also dulness over a greater area, in the region of the aorta rather than of the right ventricle, and may be further distinguished by pressure signs in producing inequality of the pulse, aphonia, dysphagia, and finally erosion of bone.

The *prognosis* is bad, chiefly on account of the extensive involvement of the heart muscle. Nevertheless, the course of the disease need not be necessarily rapid. The hypertrophy of the right ventricle may for a long time compensate the lesion. Unfortunate are the cases which complicate or are superimposed upon insufficiency of the aortic valves, so that neither the lungs nor the body are properly supplied with blo od. Any intercurrent affection of the lungs—bronchitis, catarrhal pneumonia, tuberculosis, etc.—intensely aggravates the prognosis; hence the great danger of measles and whooping cough in childhood.

The *treatment* should have special reference in prophylaxis to avoidance of exposure to the infections of childhood and to the vicissitudes of weather which excite or aggravate a "cold." The general principles of treatment are described in connection with the lesions of other valves most commonly affected in adult life.

Pulmonary Stenosis.

Pulmonary stenosis may be congenital or acquired. Stenosis of the pulmonary valves belongs among the rarest of acquired lesions, but, as observed elsewhere, is the most frequent of all the congenital defects. Most of the cases therefore are observed at birth or in early life, as the lesion becomes intensified after birth.

Congenital stenosis is nearly always associated with other anomalies of the heart, especially with permanence of the foramen ovale, of the ductus arteriosus, perforation of the septum, etc. The fact that the condition is found so often in connection with perforation of the septum is proof of the early existence of the inflammatory

process which produces the stenosis, as the septum is closed as early as the end of the second month of fœtal life. A large opening in the septum is evidence of the existence of the arrest of development at a very early period, and the presence of a perfect septum indicates the production of the stenosis at a much later period in fœtal life.

Acquired stenosis, when it occurs, is produced by the general causes of valve lesion, especially by endocarditis, but here only after involvement of other valves. v. Wahl reported an exceptional case of acute endocarditis attacking the valves of the pulmonary artery, and Bozzolo a case of vegetative endocarditis confined to the pulmonary valves though concomitant with a diffuse mural endocarditis. Paul, Woillez, Solomon, Duguet, and Landouzy all reported cases of acquired stenosis at the pulmonary orifice. Petit cites further the names of six authorities who reported separate cases, so that the development of the lesion in later life has now been abundantly demonstrated. Schwalbe further reported a case of stenosis produced by cicatricial myocarditis and syphilitic changes in the cone of the pulmonary artery. Tumors in neighboring organs more frequently produce stenosis of the trunk of the pulmonary artery or some of its main branches.

Morbid Anatomy.—The valves show the same changes as in the case of the similar lesion in the aorta, that is, the segments of the valves are usually found adherent, sometimes forming a diaphragm which shuts off the communication with the right ventricle save by a narrow orifice centrally or eccentrically situated. The lesion may vary in degree from a complete or nearly complete occlusion or perfect obliteration of the pulmonary artery, to a slight narrowing which scarcely interferes with the passage of the finger. The contraction is usually situated at the level of the valves. Vimont found the contraction at this site in twenty-two of thirty-two cases. Where the contraction lies beyond the valve in the pulmonary artery, it is usually caused by the changes of arteriosclerosis or atheroma. Elliotson speaks of cases in which the artery would scarcely permit the passage of a quill, and of other cases in which constrictions were found in various parts of its course. Chrétien collected from the literature five cases of acquired stenosis of the pulmonary artery above the ring of insertion of the valves and to these cases added a sixth of his own. In the case of the author, an old man, aged seventy-nine, who had died suddenly, a cicatricial contraction down to the size of a goose-quill was found several centimetres above the perfectly normal and competent pulmonary valves. By the side of the contraction was a small ulcer as a relic of the process which had caused it. In rarer cases the vegetations of endocarditis may be found deposited upon

the surface of the valves, or the valves themselves may be thickened and rolled up on their edges as in the case of the aortic valves. The acquired stenosis is almost never found alone; the lesion nearly always produces also an insufficiency.

Symptoms.—In the presence of an obstacle at the orifice of the pulmonary artery, the right ventricle undergoes hypertrophy and the enlargement is recognized by dulness under and to the right of the sternum, by a pulsation at the epigastrium and slight dislocation of the apex stroke. The right ventricle in these cases sometimes becomes thicker than the left. Féréol once found a thickness of 22 mm.

The accumulation of blood in the right ventricle and right auricle increases the pressure in these cavities and thus tends to prevent the closure of the foramen ovale. The same increase of blood pressure may arrest the development of the septum and leave an orifice of communication so that part of the blood is discharged from the right to the left side of the heart. The escape in this way relieves the pressure in the right ventricle but superimposes work upon the left ventricle which must now discharge an extra amount of blood. Hence it is that the left ventricle undergoes a certain degree of hypertrophy. As the condition is so often encountered in youth, the enlargement of the heart may be revealed by simple inspection of the chest, which shows a distinct protrusion.

The discharge of the blood wave is attended by a murmur heard in greatest intensity over the semilunar valves in the second interspace to the left of the sternum, and synchronous with the first sound of the heart. The murmur is usually so loud that it may be heard also in the back. According to Paul, the murmur becomes louder in dorsal decubitus than in the sitting or standing posture. It becomes fainter also under a sustained expiratory effort with the mouth and nose closed. When the patient stands and during expiration the blood pressure is raised in the pulmonary artery and this increased obstacle to the egress of blood weakens the sound of the souffle. The friction of the blood column sometimes produces a fremitus which may be distinctly perceived by palpation.

When the stenosis lies beyond the valves in the pulmonary artery the murmur may be heard in intensity in the region of the hilus of the lungs in the back near the vertebral column. The second sound is faint or fails entirely. In the presence of a coincident insufficiency the second sound is substituted by a diastolic murmur.

As remarked elsewhere, the congenital cases are usually associated, not only with defects in the heart, but with arrest of development of the whole body.

Congenital stenosis is attended with cyanosis, which is often

extreme, but the associate defects, permanence of the foramen ovale and of the ductus arteriosus, to some extent compensate the lesion by relieving the right ventricle of the extra work imposed upon it. These defects, together with the hypertrophy of the right ventricle, may compensate the lesion for a long time. Most of these cases show also general arrest of development of the rest of the body, including the intellect. The subject of pulmonary stenosis shows prominent eyes and thick, red lips. The veins of the neck, chest, and extremities are distended and form a visible anastomosis. The thorax is narrow, the precordial region is prominent and is agitated with violence under the impact of the heart; the abdomen protrudes under the enlarged liver; there is defective development of the genital organs; the fingers and toes, strongly cyanotic, show clubbed extremities (drumsticks) and big curved nails. Attacks of dyspnœa, suffocation, hæmoptysis, epistaxis, and convulsions are not infrequent. The subjects of pulmonary stenosis are also liable to headache, hebetude, somnolence, and insomnia, with subjective and objective sensations of cold. But there is much less tendency to dropsy than in other valve lesions.

Pulmonary stenosis has always been considered a predisposing cause of tuberculosis in contrast with other valve lesions, which are said to confer a certain degree of immunity. The attempt has been made to explain these differences by the fact of a defective blood supply in cases of obstruction of the pulmonary artery. Probably the defective development of the whole body, including hypoplasia of the lung tissue itself, may have more to do with the disposition to these diseases. Contracted apices may long retain, and defective ciliary action may fail to extrude, the tubercle bacilli, which find also a favorable nidus for development in consequence of the defective nutrition.

Diagnosis.—In the presence of a congenital lesion this is not difficult. The cyanosis, which is intensified by either coughing or crying, and the increased size of the heart, manifest in the vaulting of the chest and more powerful action, in association with the characteristic defects of the body, may leave little room for doubt.

Pulmonary stenosis must be separated from cases of permanent foramen ovale which may furnish many of the same signs, especially the cyanosis. The distinction is important because the permanent foramen ovale is not incompatible with long life, and is, therefore, a much less serious lesion. The signs of pulmonary stenosis are usually more grave, especially is the dyspnœa much more severe. Moreover, the permanent foramen would fail to show the physical signs of pulmonary stenosis: hypertrophy and dilatation, fremitus and murmur.

The diagnosis is more difficult in the acquired cases. The region of the pulmonary valves is famous for the number and variety of its murmurs. Guitéras distinguishes this region as "romantic" in this regard. Diastolic murmurs more distinctly refer to organic lesion of the pulmonary valves. Systolic murmurs arise from many causes independent of valve lesion. The frequency of murmurs in this region has been ascribed to the close apposition of the pulmonary artery to the wall of the thorax and the facility of compression of the delicate wall of the vessel by light pressure. An accidental murmur is often heard in young strong people in the region of the pulmonary valve. Moritz finds in this case a systolic murmur which is longer in expiration and which has usually been referred to compression of the pulmonary artery by mediastinal gland tumors. This author encountered the condition in two young, perfectly healthy men in whom the murmur disappeared entirely in inspiration, but was very loud in forcible expiration. The following conditions, he finds, are necessary to the development of it: A flat, very elastic thorax, thin springy ribs, expansible lungs which withdraw themselves from the heart during expiration so that a large surface of the heart being uncovered is apposed to the thorax; a large excitable heart; strong chest muscles. Under these conditions the pulmonary artery is compressed by the wall of the chest during expiration and a murmur is developed in this way in the entire absence of any abnormality. Of course tumors and infiltrations may produce the same effect. The influence of respiration by increasing the blood wave may also furnish favorable conditions for vibrations (Rosenbach). It is only in the rarest cases that a murmur indicates a lesion of the pulmonary valves, and the diagnosis may be justified only when there is reason for the lesion in the preexistence or coexistence of other valve lesions, and when the physical signs array themselves in order. Claisse reported a case in which a group of tuberculous lymph glands behind the sternum was the cause of a systolic murmur which had been erroneously considered as a stenosis of the pulmonary orifice.

Cyanosis alone by no means of necessity indicates pulmonary stenosis, as this symptom may occur in any lesion of the heart, and the various defects of development may also be found in connection with other lesions or in the entire absence of affection of the heart, as in congenital anomalies of the lungs, early emphysema, asthma, etc. It is needless to state that cases of pulmonary stenosis may show no defect in development and no cyanosis whatever throughout the whole course of the disease. The differences which exist in different individuals are extreme in this regard and are explained with difficulty on anatomical grounds.

The *prognosis* is bad, but depends upon the existence of the lesion, the character of the complications with other valves, and the condition of the heart muscle, together with the general nutrition or development of the body. As a rule, patients affected with pulmonary stenosis do not survive adolescence, and only the most exceptional cases reach maturity and old age. Death occurs most frequently in some attack of cardiac asthma or convulsion; or the patient suffers the slow marasmus of catarrhal pneumonia and tuberculosis. It is questionable if any comfort can be given to the patient on account of an easier compensation through a coexistent defect in the septum of the auricles or ventricles.

The prognosis and treatment of pulmonary stenosis have been studied in detail in the section on Congenital Anomalies.

Combined Valve Lesions.

The various lesions of the valves may be associated in every kind of combination. This association is usually a sequence, as one valve after another is affected in the course of the original and originating malady, or more frequently in the course of relapses, recurrences, or subsequent attacks. As already frequently remarked, it is rare that a lesion so affects a valve as to disturb the function in only one way—that is, it is rare to find an absolutely pure insufficiency or stenosis. The same lesion produces both conditions, but in varying degree, so that one or the other disturbance predominates. The rule is, the more distinct the insufficiency the more indistinct the stenosis, and *vice versa*. In a general way it may be said that insufficiencies more frequently than stenoses exist alone, *i.e.*, an insufficiency may be pure, but an obstruction nearly always implies the coexistence to some degree of regurgitation.

Sometimes the disease spreads by contiguity of structure. Thus the aortic valves may become affected from the aortic side of the mitral valves. So the process of atheroma may extend from the aortic valves to affect the mitral valves. When the mitral valves are closed the distance between the two sets of valves is not great. Sometimes the valves are affected by their physiological connection. Thus insufficiency or stenosis of the mitral valve leads in the course of time to insufficiency of the tricuspid valve. An aortic or pulmonary insufficiency, by leading finally to dilatation of the left and right ventricle, will eventually bring about relative insufficiency of the mitral and tricuspid valves. Stenosis at the aortic and pulmonary orifices may bring about relative insufficiency of the mitral and tricuspid valves. Stenosis at the aortic and pulmonary orifices may bring

about the same relative insufficiency even sooner, in consequence of the earlier exhaustion of the heart under the processes of arteriosclerosis.

But aside from these combinations of organic lesions with relative insufficiency, great caution should be exercised in the diagnosis of combined lesions. Notwithstanding the apparent evidence to the contrary, at the autopsy there is usually revealed an affection of but one set of valves, and the confusing symptomatology is found to be due to relative insufficiency.

Insufficiency and Stenosis of the Mitral Valve.

Insufficiency of the mitral valve with stenosis at the left auriculoventricular orifice constitutes the most frequent of the conjoined lesions. As a rule, the insufficiency predominates. In much rarer cases the stenosis is the larger lesion. In these cases the condition is distinguished by the more rapid dilatation of the left auricle, more intense accentuation of the pulmonary valve sound, which may even be reduplicated, and by the speedier development of hypertrophy and dilatation of the right ventricle. Auscultation reveals a presystolic (or diastolic) as well as a systolic murmur, both heard in greatest intensity at the apex of the heart. The progress of the disease is more rapid in every way, as the condition partakes of the graver lesion, to wit, the stenosis at the left orifice.

Insufficiency and Stenosis of the Aortic Valve.

The same qualifications pertain to obstructive lesions in regard to regurgitation at the aortic orifice as in the case of the auriculo-ventricular orifice—that is, the lesions are not always pure. But insufficiency of the aortic valves is found much more frequently alone than is that of the mitral valve. It is known, however, that the conditions may coincide and one or the other may not be recognized. This is especially true in the case of insufficiency. As Guttmann has already pointed out, in aortic stenosis of high degree the diastolic murmur may be entirely absent. A predominant insufficiency may also conceal a light degree of stenosis. Moreover, a systolic murmur in the region of the aortic valve may depend, not upon a stenosis, but upon endarteritic changes in the ascending aorta. In the conjoined lesion the pulse shows the combined characteristics, the small, hard, wiry pulse of stenosis with the bounding character of insufficiency. The double lesion is disclosed by a double murmur, systolic and diastolic, both heard in greatest intensity at the base of the heart.

Stenosis of the Mitral Valve with Insufficiency of the Aortic Valve.

This combination is not infrequent, and is found in connection with the great hypertrophy of the left ventricle, which makes itself manifest by the increased impact of the whole heart, and by the presence of two murmurs, both coincident with the systole, one heard in greatest intensity at the apex and the other at the base. A. H. Smith has modified the double Cammann stethoscope, with separable chest pieces, one of which might be applied at the apex and the other at the base, so that variations of intensity in the sound at these regions may be simultaneously appreciated. Such an instrument may furnish valuable evidence, but could give positive information only in those cases where the acuity of hearing was exactly the same in both ears, which is seldom the case.

Stenosis of the Aortic Valve with Insufficiency of the Mitral Valve.

This combination, which is quite uncommon, would be revealed by a lesser degree of hypertrophy of the left ventricle, by a predominant dilatation of the left auricle and hypertrophy of the right ventricle, and by the existence of two bruits, both systolic, one heard in greatest intensity at the apex, the other at the base, in the second interspace to the right of the sternum.

Insufficiency of Both Mitral and Aortic Valves.

In this combination the aortic valves are most frequently affected with endocarditis, while the mitral valves are only relatively insufficient. The left ventricle is usually found in a state of hypertrophy or dilatation. There are two murmurs: one is heard in greatest intensity at the apex, synchronous with the first sound of the heart, in consequence of the insufficiency of the mitral valve; and the other is heard in greatest intensity at the base, synchronous with the second sound, in consequence of insufficiency of the aortic valves. As systolic murmurs are so frequent, the diagnosis of a double lesion of this character may not be justified except in the presence of accentuation of the pulmonary valve sound. Botkin, Saundby, and Timofejew have all remarked upon the fact that the murmur of aortic insufficiency may be entirely absent when the mitral insufficiency is extreme.

Stenosis of Both Mitral and Aortic Valves.

Lesions which produce an obstruction at the aortic orifice and left auriculo-ventricular orifice are rare. In such a case the mitral stenosis is indicated by the rapid dilatation of the left auricle, with the prominent signs of pulmonary stasis, while the aortic stenosis is shown in the defective blood supply to the various organs, especially to the brain and kidneys. There is, in consequence of mitral stenosis, hypertrophy and dilatation of the right ventricle, and in consequence of the aortic stenosis some degree of hypertrophy in the left ventricle. There are also two murmurs: that of mitral stenosis, presystolic or diastolic, heard in greatest intensity at the apex; and that of aortic stenosis, heard in greatest intensity at the base and synchronous with the first sound of the heart.

Besides these combinations of valve lesions of the left side of the heart, there may be coexistent affection of the valves upon the right side of the heart. As repeatedly remarked, the organic valve affections upon the right side of the heart belong chiefly to uterine life. The practitioner will therefore make a diagnosis of organic defect of the tricuspid or pulmonary valves only with the most extreme caution and reserve. So far as the right side of the heart is concerned, the condition most frequently encountered is a relative insufficiency of the tricuspid valves, which may occur in connection with any lesion of the heart, but is found most frequently in association with affection of the mitral valve.

Tricuspid Insufficiency with Lesions of the Mitral Valve.

Insufficiency of the tricuspid orifice occurs in connection with both lesions of the mitral valve, most frequently in connection with insufficiency, because insufficiency is the most frequent lesion, but most rapidly with stenosis. In addition to the signs indicative of the affection of the mitral valve, tricuspid insufficiency is indicated, as stated, by the rapid increase of dulness to the right of the sternum on account of dilatation of the right auricle, as well as of the right ventricle, by the signs of general stasis, dropsy, and cyanosis. The murmur of tricuspid insufficiency may be distinguished from that of mitral stenosis by the period of its occurrence, as that of mitral stenosis is presystolic or diastolic; but the murmur of tricuspid insufficiency cannot be separated from that of mitral insufficiency, which it really only intensifies or prolongs. The diagnosis

is then established by the signs of venous stasis, by the venous pulse in the neck, by the liver pulse, etc.

Tricuspid Insufficiency with Insufficiency of the Aortic Valves.

This combination is much less frequent because of the predominant and sustained hypertrophy of the left ventricle, which compensates the lesion of the aortic valves. When it occurs, however, it may be recognized by the conjunction of symptoms which belong to the two conditions. Thus the aortic insufficiency is distinguished by the great hypertrophy of the left ventricle and powerful impact of the heart, also by the peculiar "pistol-shot" pulse and by the capillary pulse with the double tones and murmurs in the crural arteries; whereas, to the tricuspid insufficiency belong the signs of venous stasis and interference with circulation in the lungs, cyanosis, and dropsy.

Tricuspid Stenosis Combined with Other Valve Lesions.

This combination, though rare, occurs most frequently in connection with stenosis at the mitral orifice. Trekaki remarked upon this frequency of the simultaneous occurrence of mitral stenosis and tricuspid stenosis. In the collection of combined lesions by Leudet, this condition was observed in seven-eighths of the cases.

That these various combinations do occur, and that they are sometimes accurately diagnosticated in life, may be seen in the citation of a few cases from competent authorities. Thus:

Maximowitsch described the history and pictured the pulse curves with cardiograms of four cases affected with mitral stenosis and insufficiency and aortic stenosis and insufficiency. In all four cases the complication, diagnosticated during life, was confirmed by section. The diagnosis was based upon the disseminated murmurs about the heart synchronous with the sounds or substituting the sounds, the hypertrophy of both valves of the heart, arhythmia of the pulse, whose individual waves resembled the pulsus celer, and absence of other complications. Von Ziemssen reported a case of insufficiency and stenosis of the aortic with insufficiency and stenosis of the mitral valve; Barr a case of aortic and mitral stenosis with large calcareous mass in the posterior cups of the mitral valve and with commencing stenosis of the tricuspid orifice; Bellingham a case of cribriform condition of the aortic and pulmonary valves; Hayden and Horrocks a case of incompetent mitral and tricuspid stenosis;

Irvine a case of mitral and tricuspid stenosis; Lepine a case of extreme contraction of the right auriculo-ventricular orifice coincident with an extreme coarctation of the left auriculo-ventricular orifice; Carrington a case of mitral and tricuspid stenosis with concurrent disease of the aortic valves; Clinton a case of ossification of the mitral and aortic valves with induration of the tricuspid valve, and hypertrophy and dilatation of both auricles, while the ventricles were sound. Middleton, Thatcher, and Drummond described each a case of extreme tricuspid stenosis in connection with stenosis at the aortic and mitral orifices. In the case of Middleton it was remarkable that stasis in the lungs was entirely absent, notwithstanding the grave disturbance of compensation. The explanation was found in the fact revealed upon autopsy that the tricuspid orifice was changed to a round opening two inches in circumference. In the case of Drummond there were no symptoms which could be referred to implication of the tricuspid valve other than the fact that the presystolic murmur of mitral stenosis could be heard further to the right than usual. Trekaki demonstrated to the Paris Anatomical Society the heart of a twenty-six-year-old woman, affected with mitral stenosis of high degree and stenosis of the tricuspid orifice. The left ventricle was markedly hypertrophied, but there was no hypertrophy in other parts of the heart. During life there was observed the fremitus câtaire and a presystolic râle, but there was no reduplication of the second sound. Finally, Grawitz reported the case of a man aged twenty-four who had suffered for five years with grave symptoms of stasis and had shown numerous heart murmurs. The diagnosis had been established as stenosis and insufficiency of the mitral valve, with endocarditic alterations in the aorta, insufficiency of the tricuspid valve, and stenosis at the right auriculo-ventricular orifice. The presence of a palpable systolic and diastolic fremitus at the fifth costal junction on the right side in connection with a very loud murmur in the region of the tricuspid valve, were the signs upon which the diagnosis was based. The autopsy confirmed the diagnosis in every respect. In another case a man aged forty-three had suffered twenty-two years from rheumatism and mitral insufficiency, and stenosis had set in. In the course of time there developed, further, strong pulsation and diastolic fremitus under the ensiform cartilage, which was referred to the tricuspid valve. There could now be heard over the pulmonary artery a soft systolic and a long-drawn diastolic murmur. The right border also of the lungs was depressed and immobile. Inspiration was effected in two to three detached efforts. At the time the report was made the patient was still under treatment and was in good general condition.

General Diagnosis of Valve Lesions.

The diagnosis of chronic valvular disease is usually a matter of little difficulty. Many cases may be latent for a long time. These cases remain undiagnosticated more for the reason that no symptoms point to the heart. In other cases the signs of disease refer the practitioner rather to some other organ, more especially to the lungs or to the brain, stomach, kidney, or other organ affected by the disturbance of circulation or suffering under defective nutrition. In the majority of cases the patient does not complain of the heart, and the symptoms are not of the character which are commonly believed to belong to disease of the heart. Most of these symptoms, to wit, palpitation, pain, and anxiety, belong really to affection of other organs or are evidence of disturbance of innervation rather than of organic disease of the heart itself. Patients who complain of heart disease usually suffer with some of the neuroses. In the majority of cases the diagnosis is determined, not by the subjective signs, which are vague and indistinct, but by the physical signs, which are exact and positive. But the genetic relation of previous affection may lead to an examination of the heart and thus establish a diagnosis. Thus the connection between rheumatism and heart disease has become so well recognized as to make the patient himself suspicious of this complication in every attack. Very often, however, the fears of the patient are allayed while nevertheless a chronic endocarditis is in process of insidious development. In the majority of cases chronic endocarditis develops in this way. When the existence of the disease is appreciated by the physician for the first time, the patient is able to recall, sometimes only under searching examination, the previous existence of an attack of rheumatism, pneumonia, typhoid fever, influenza, or other infection. It is only in the minority of cases that the chronic endocarditis develops in connection with a recognized acute form of the disease.

The diagnosis really rests upon the physical signs, and the most reliance is placed upon the enlargement in the diameters of the heart, which indicates hypertrophy or dilatation. Any marked increase in the outlines of the heart is usually readily recognized. The clinician is at once impressed with the dulness and flatness in regions which should yield the resonance of the lungs. But, as stated and emphasized, hypertrophy may be distinct, as evidenced by the general signs, or as proven on autopsy, and may fail to reveal itself under the coarser means of study by percussion. So much depends upon the situation of the lungs with reference to recession or advance, with

reference to intercalation between the heart and chest, with reference also to the degree of expansion of the air cells, states of emphysema, etc. It is interesting, therefore, to know that a new means for the detection of hypertrophy has been offered in the more delicate appreciation of the heat test. Meissner maintains that the skin over the heart is warmer than that over the lungs (not colder, as was claimed by Benezúr and Jónás). The difference may not be apprecitated by the touch, but can be clearly recognized by a suitable apparatus, so that the limits of the heart may be distinctly noted. More than this: the right ventricle gives out more heat than the left, and Meissner succeeded in several cases in defining the situation of the ventricular septum in the medical clinic at Göttingen. Thus Piorry, who is said to have been able to map out the position of the placenta on the wall of the uterus, is surely surpassed even in any dreams that he might have entertained regarding the perfection of percussion.

As the outlook depends really upon the condition of the heart muscle, the factor of utmost importance is the recognition of dilatation. This factor is usually revealed by diminution in the force and increase in the frequency of action of the heart and of the pulse. Cases of distinct dilatation are always marked by tachycardia. Finer changes may be recognized by alterations in the sounds of the heart. Thus Broadbent finds that a relatively soft first and relatively loud second tone at the apex speak for the presence of an hypertrophy. Impurity of the first tone, caused usually by reduplication, speaks for weakening of the left ventricle (dilatation) which is working against abnormal obstruction. Retardation of the interval between the first and second tones signifies hypertrophy. Curtailment of this interval is a sign of defective emptying of the ventricle and signifies dilatation.

The stage of full compensation is indicated by the fact that, notwithstanding the lesion, the action of the heart suffices for the wants of the body. This stage is coarsely indicated by the condition of the pulse. Compensation begins to be broken as soon as the blood pressure in the arteries is permanently reduced. In the case of insufficiency of the mitral valves, compensation is broken when the right ventricle fails to overcome the dilatation in the left auricle and thus permits accumulation in the auricle and lessens the quantity of blood delivered to the ventricle and to the aorta; and in the case of insufficiency of the aortic valves, compensation is broken when the left ventricle fails to discharge into the aorta the normal amount of blood. In lesions of the tricuspid and pulmonary valves compensation is broken when blood begins to accumulate in the lungs and in the

venous system. Liebermeister makes a good point when he regards the temperature of the periphery as a criterion of the course or rapidity of the circulation. The temperature of a peripheric part of the body, in comparison with the temperature of the interior and that of the vicinity, furnishes a criterion for the circulation, so far as this part is concerned. In bad cases of valve lesion the surface is cold as well as blue.

Accidental Murmurs.—In the interpretation of murmurs the so-called accidental murmur must be disposed of first. The accidental murmur is usually soft and more distinctly blowing. The accidental murmur belongs chiefly to states of anæmia and cachexia. It is heard most distinctly over the base of the heart and in greatest intensity in the region of the pulmonary valves; it corresponds with the systole. A diastolic murmur is almost never accidental. But systolic murmurs in aortic insufficiency are sometimes due to roughnesses produced by arteriosclerosis in the aorta itself in the entire absence of affection of the valves. Then there is no doubt also that diastolic murmurs are sometimes accidental, that is, independent of valve lesion. These murmurs may be heard especially in the anæmic. Thus Sahli found diastolic accidental murmurs at the apex in the case of two women affected with pernicious anæmia. The autopsy disclosed no lesion and it was seen that relative insufficency could not be established. Sahli believed that these murmurs developed in consequence of extreme hydræmia, as they became most intense toward the end of the disease. On this account the murmur could not be attributed to a greater increase in the circulation. The diastolic murmur had also nothing to do with the accidental systolic *bruit de diable* present at the same time. Further, pericarditis, sclerotic surfaces, or embolic spots on the pericardium may give rise to pure diastolic murmurs at the base of the heart (Gerhardt). Finally, a diastolic murmur may be heard in aneurysm of the aorta.

Moreover, venous murmurs arising in the veins of the neck (Sahli) or in intrathoracic trunks (Duroziez) may be conducted to the heart and simulate valve murmurs. Crook found accidental murmurs in the vessels in 51 of 1,500 persons; of these cases 42 were venous and 26 arterial murmurs. The majority of the individuals were anæmic, and of the anæmic 90 per cent. show murmurs in the vessels. Richardson found venous murmurs in one-half of his chlorotic patients in whom the hæmoglobin was reduced 50 per cent. The murmurs were audible only on the right side in 33 per cent., only on the left in 6 per cent., and on both sides in 11 per cent. of the cases. These murmurs always disappear with increase of the hæmoglobin. The systolic heart murmur is, however, more obstinate and yields only

when all the other symptoms of chlorosis have entirely disappeared. Accidental murmurs vary more. They may be dissipated by toning the muscle of the heart or improving the condition of the blood. Audeoud and Jacot-Descombes, in a prize essay submitted at Geneva, describe a functional mitral stenosis in nervous, irritable people, affected for the most part with chloro-anæmia. These cases show a presystolic, sometimes a diastolic murmur, duplication of the second sound, presystolic fremitus, etc., in the entire absence of endocarditis. These authors assume in explanation of the condition a spasmodic contraction of the valve ring or of the papillary muscles, which narrows the auriculo-ventricular orifice and thus produces the symptoms of mitral stenosis. They reported twelve cases in illustration of the condition, and remarked upon the fact that in all the cases the auscultatory phenomena were very variable. In two cases which succumbed to other diseases there was found more or less pronounced chronic nephritis. At the same time it must be admitted that many, perhaps most, accidental murmurs depend upon relaxation of the heart muscle, weakness of the papillary muscles, failure of adjustment of the orifice, producing or permitting a relative insufficiency with reflux of blood, and eventually with some degree of dilatation. A typical example of such a cause is seen in the action of beri-beri which quickly poisons the heart muscle. Curnow found the heart dulness in his cases at the Seaman's Hospital at Greenwich markedly increased to the left, and heard murmurs of relative insufficiency distinctly audible at the apex and over the aortic and pulmonary orifices, which disappeared later under rest and digitalis. The pulse was irregular, arhythmic, often 120 and more.

The organic murmur occurs in a case marked also by hypertrophy and by accentuation of other valve sounds.

The true murmur of organic disease stands in definite relation to the different phases of the action of the heart. In this way an endocardial murmur may be separated from a pericardial friction sound. Pericarditis may leave rough surfaces and furnish friction sounds which may closely simulate endocardial murmurs. Pericarditis with effusion is positively recognized by aspiration. Cicatricial thickening may embarrass the diagnosis. It is observed, however, that the friction sound of pericarditis does not correspond so definitely to the phases of the heart's action. They may be altered by change of posture of the patient, by pressure with the stethoscope, etc. The pericardial friction sound from cicatricial thickenings is usually systolic, but shows no accentuation of the sound of the pulmonary valves and—aside from effusions—need not be associated with change in the diameters of the heart.

As may be readily understood, the natural sounds of the heart are best appreciated when the action of the heart is slow. Where the interval between the systole and diastole is very short, the first and second sounds seem almost to run together. Unnatural sounds or murmurs are likewise easiest appreciated under slow action of the heart. Unfortunately in many cases the disease which produces the murmurs accelerates the action of the heart. This is especially true in acute stages of the disease. It becomes again true in the stage of failing compensation, and in the last stages of heart failure it may be entirely impossible to distinguish anything clearly. It is so also with the regularity of the action of the heart. Any irregular action, arhythmia, adds to the difficulties of diagnosis. Under slow and regular action even a feeble murmur may be distinctly made out. Under tumultuous and irregular action all the sounds become confused, and it is a fact that practised auscultators are guided in complex cases more by feeling and certain acoustic memories than by any objective perception of conditions of momentary duration (Vierordt).

The sitting or reclining posture distinctly intensifies murmurs by retarding the pulse. The "position relevée" recommended by Azoulay, is especially valuable in this way. In this position the body lies horizontally, the chin is brought in contact with the chest, the arms are held vertically; the lower extremities are likewise elevated, or at least the heels are brought up to the pelvis. This position intensifies heart murmurs in maximum degree in cases of valve lesion, while it has no essential influence upon chronic myocarditis. The true pericardial murmurs are most distinct in a position which is upright or lightly inclined forward. Gerhardt maintains that murmurs which are absent while lying, are developed while standing, in beginning insufficiency of the aortic valves, while the reverse is true in beginning insufficiency of the mitral valves.

The diagnosis is sometimes established suddenly in the processes of embolism. Thus the sudden occurrence of aphasia or hemiplegia in a young person previously affected with rheumatism, may leave little doubt as to the nature of the lesion.

Hyperæmia of the retina has sometimes been observed in the course of valve lesion. The ophthalmoscope shows the veins distended and dilated in this condition. Diedreich speaks of the cyanotic retinitis as accompanied by light serous transudation. Galezowski found the retina often covered with numerous red points of extravasated blood. Embolism of the central artery of the retina is announced usually suddenly sometimes in consequence of fright or some mental emotion and in the absence of any pain or sign of in-

flammation (André). The ophthalmoscope in these cases shows the papillæ anæmic, while the arteries, reduced to threads of extreme tenuity, traverse the retina as white lines.

The diagnosis may become difficult in the case of embolism in the lungs, where the symptoms may simulate pleurisy, pneumonia, bronchitis, or even tuberculosis. Embolism of a chief or large branch of the pulmonary artery reveals itself in signs of unmistakable gravity, dyspnœa, anxiety, convulsions, etc. Infarction of smaller vessels is attended by pain and is more distinctly specified by the sputum, which is bloody and which contains blood corpuscles with pigment cells, amorphous or in the form of needles or rhombic tables, partly free and partly included in the cells, the so-called cells of heart failure. The condition is distinguished from pleurisy by the absence of any sputum in pleurisy, and usually by the absence in infarction of a friction sound, which furnishes such distinctive evidence of pleurisy. Croupous pneumonia, which is the only pneumonia of sudden occurrence, is distinguished by its initial chill, by its high temperature, and by its range of infiltration.

As the immediate future of a case depends really upon the condition of the heart muscle, the factor of supreme importance is the recognition of the disturbance of compensation. As so often remarked, it is of far more value to know the condition of the heart muscle than to be able to locate exactly, or even determine the extent of, a lesion of the valves. As will be seen, most cases remain stationary for a long time, for months or even years; many cases for even decades of years; sometimes the symptoms are so latent that the disease is concealed. But in all cases the lesion is revealed sooner or later by the condition of the heart muscle. The heart must, under all circumstances, continue to do its work, and whether more or less will depend upon the surroundings of the patient, the habits of the individual, the strength of the will, etc. The heart muscle draws more and more upon its reserve force. Age advances. There may be intercurrent disease. Any disease of any organ makes extra demand upon the heart, but these demands are especially felt in the course of intercurrent affection of the lungs, bronchitis, asthma, emphysema, pleurisy, pneumonia, etc. No life is free from emotional disturbance, which makes extra demands upon the heart. Then, it is not the nature of the disease process itself to stand still indefinitely. Other muscle fibres become successively involved. Finally, there is failure in compensation. The point in diagnosis which is supreme is the recognition of the first failure in compensation, that the condition of life may be changed, that the demands for superfluous work may be removed, that the heart muscle

may be toned and strengthened for the work which is absolutely necessary to life.

Failure in compensation is inevitable. What are the first signs of failing compensation? The physician who is familiar with the disease in the individual, with the habits of the individual, and who has the confidence of his patient so that the patient consults him from time to time, will be on the lookout and will discover these signs early.

The signs of failing compensation have been mentioned in speaking of the early symptomatology of valve lesions. The earliest signs are the symptoms which are first seen in the most insidious and so-called latent cases. Some of these signs do not point directly to the heart as the seat of the disease. They indicate rather a general failure of health and strength, and in many cases, as stated, the diagnosis is only reached by an examination of the whole body, which discloses a lesion of the heart. For, aside from the physical signs, the heart lesion may show no symptoms until the heart muscle begins to flag in its force, and as the force of the heart varies the symptoms at first are not continuous but show themselves only from time to time.

Now, the character of the first symptoms will depend upon the situation of the lesion, whether the mitral or aortic valve be affected first or most. When the mitral valves are affected the evidence of failure is first found in the lungs; when the aortic valves are affected the evidence of failure is first found in the systemic circulation. Later it is seen that interference with one set of valves disturbs the action of the other, so that final effects are the same. Failure of compensation has reference to the beginning of this later stage, when the blood is no longer driven through the lungs in sufficient force or quantity to secure full aeration of the blood, and through the aorta with sufficient power to supply the wants of the body. A failing force of the heart is therefore seen first in the falling of the blood pressure in the blood-vessels, and this lowering of pressure may be appreciated soonest by the use of delicate apparatus, arteriometer, sphygmograph, etc., which furnish evidence of value, however, only when the lowering of pressure is found to be more or less continuous. Such apparatus can be adjusted, of course, only to the systemic vessels, and is practically limited in application to the radial artery.

But observation often accurate enough for all practical purposes can be made in the ordinary study of the pulse, whereby it is seen that the force and rhythm of the action of the heart are changed.

The interference with the circulation of blood through the heart, which leads to the lowering of blood pressure in the arteries, leads in like manner to an increase of the pressure of blood in the veins.

The evidence of this change of blood pressure is furnished first in the lungs. As so often remarked, the distention of the capillaries in the lungs, with retardation of circulation, interferes with aeration of the blood, and brings about changes of nutrition in the epithelium of the air cells themselves, which interfere with the diffusion of gases and the general processes of metabolism in the lungs. Hence the individual soon feels the *besoin de respirer* (want of air). The want is felt at first only under extra demand, especially from muscular exercise, but later shows itself even in states of perfect tranquillity, and amounts in the course of time to a veritable *Lufthunger* (hunger for air), which shows itself finally in the terrible struggles, the almost convulsive manifestations of cardiac asthma.

Under the diminished supply from the arteries and the increased pressure in the veins, the skin loses its color, becomes pallid and later smoky or dusky. Still later, as the defect in aeration becomes extreme, venous blood accumulates in the capillaries and reveals itself by the peculiar bluish discoloration, first about the lips, later in the extremities, the finger-nails, and finally in the whole face, known as cyanosis. In marked cases the cyanosis extends from the feet to the legs, which become swollen, cold, and blue.

Even before this time the accumulation in the veins is seen in the distention of the vessels in the neck, which in emaciated individuals stand out finally as blue-black cords, throbbing and pulsating with every action of the heart, that is, with the different actions of the right auricle and right ventricle. The same defective supply of arterial blood and increase of venous blood is felt in all organs of the body, but is perceived, as stated, especially early and distinctly in the brain, liver, spleen, and kidneys. Prominent among the signs of earliest failure in compensation are loss of energy, difficulty of concentration of ideas, insomnia, or somnolence, headache and hebetude, sometimes vertigo, as indications of the disturbance of circulation in the brain.

A sense of fulness or pain from distention of the capsule, depression of spirits, with tints of jaundice in the conjunctiva from absorption of bile in consequence of catarrhal states of the bile ducts, anorexia, vertigo, disturbed nutrition of the cells which now permit the accumulation of toxins, show in the general phenomena of "biliousness" the disturbance in the circulation of the liver. Pain in the left hypochondrium with a sense of heaviness and weight indicates swelling of the spleen and distention of its capsule. The urine is scanty and high-colored, is sometimes albuminous, and sometimes it shows hyaline casts, as early signs of a defective blood supply to the kidneys. In all these organs the accumulation of toxins leads

to exuberance of connective tissue with subsequent shrinkage and cicatrization, constituting the process of cyanotic induration. In this condition any of the organs, especially the liver and spleen, are found to be distinctly enlarged beyond their natural outlines.

General Prognosis.

The prognosis in a case of valve lesion is determined by such a multitude of factors as to render invalid the application of any general rules to individual cases. As already repeatedly intimated, lesions of the valves of the heart are by no means of necessity fatal. We may go further and say that valve lesions are by no means incompatible with long life. These facts are demonstrated by the long periods of latency of many cases and by the discovery upon the post-mortem table of lesions which gave no signs in life. In this connection may be quoted for the last time the remarkable experience of Sir Andrew Clark, who reported six hundred and eighty-four cases of chronic valvular lesions which had remained under his own observation for five years without the least disturbance of the general health, so far as the heart was concerned. Balfour speaks of the acquaintance of a hale old gentleman, eighty-six years of age, who for sixty-six of these years was known to have suffered with a dilated and hypertrophied heart. Sixty-six years, he says, is certainly the longest period in his experience that mitral regurgitation has been known or even surmised to exist. Many even more remarkable cases may be found in the literature. One of the most remarkable was a case reported by Carroll of congenital malformation of the tricuspid valve with lesions of all four valvular orifices; the patient survived for twenty years with few heart symptoms during life.

It is a mistake also to suppose that even organic lesions of the heart are necessarily permanent. They certainly do remain, as a rule, but in exceptional cases all signs of the lesion may disappear or what evidence remains may be changed for the better. Sometimes, as stated, the thrombus which caused the interference with the circulation has been washed off from the valves of the heart. More satisfactory are the cases in which a valve defect is relieved by change in another valve. Thus the aortic valve is highly elastic and extensile, and it happens not infrequently that the deficiency which would be left by the shrinkage of one or two valves is compensated or partly closed by the enlargement of the third valve. Sometimes the agglutination or adhesion of two valves may cover the defect of a great lesion. In exceptional cases an insufficiency may be counteracted by the elongation, for instance, of a papillary muscle.

Now and then it happens that a valve lesion becomes an escape lesion and is actually salutary. Thus Traube reported a case of sclerosis of the aortic valves with the pains of angina pectoris, in which relief of the difficulties was secured in consequence of insufficiency of the mitral valve which was attended by hydrops; and Thomson reported a case of double mitral and aortic regurgitant murmurs with marked relief from the symptoms. Musser declared that he had several cases in which the occurrence of dilatation, and the development of a mitral regurgitant murmur, in persons who had previously had hypertrophy of the heart, due to vascular changes, gave relief to the cardiac symptoms, to the violent palpitation, precordial distress, and in each instance to attacks of angina. One patient, who had an aortic lesion, had a very severe attack of angina extending over a period of four or five weeks, so that her life was despaired of, but the development of the mitral murmur relieved her of pain and other cardiac symptoms. Exemption from attack had now lasted four years. Recently other cases had occurred in which similar relief had been secured to cardiac symptoms and high tension, with which was associated headache and congestive symptoms referable to the brain, when the mitral murmur developed, the murmur being due undoubtedly to dilatation. "This has occurred to me so often that I have been rather glad to welcome the development of the murmur of mitral regurgitation or dilatation in individuals who have had symptoms due to arterial tension and to the consecutive hypertrophy of the heart."

But relief of this kind is rare. As a rule the condition persists, remains stationary at least, and in many cases, in fact, in most cases, becomes progressive. The prognosis *quoad vitam* may not be bad, but the prognosis *quoad valitudinem completam* is always bad. The disease process advances in consequence of new attacks of some infection, especially of rheumatism, whose cause remains concealed in the body, probably as a cryptogenetic sepsis, and invades the blood from time to time after exposure to some exciting cause, or independent of any such exposure. Thus the action of the heart becomes additionally crippled by extension of the disease process on the same valves or by implication of new valves. In other cases the disease spreads because it is due to affections which are in their nature continuous and progressive—for example, arteriosclerosis, atheroma, Bright's disease, etc.

When valve lesions were first recognized, they were looked upon as necessarily fatal. The reaction from this gloomy view in the course of time became extreme, with the tendency to underrate the gravity of these affections. What gives a case gravity is the fact that the

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same affection which produces the endocarditis produces also a myocarditis, and myocarditis is by nature progressive in the same sense, though slower perhaps—for the processes are not exactly alike—as in the case of cirrhosis of the kidney, of the liver, of the brain, etc.

But something of the prognosis may be determined from the nature of the lesion. Thus, as already repeatedly remarked, diastolic lesions are graver than systolic lesions. The most favorable of all valve lesions is mitral insufficiency, next comes insufficiency of the aortic orifice, third stenosis of the aortic orifice, while mitral stenosis has a prognosis almost as bad as the lesions of the right heart.

The prognosis of relative aortic insufficiency is bad, because the heart muscle is weak and the aorta itself is affected often with arteriosclerosis. On the other hand, in relative insufficiency of the mitral valve the prognosis is favorable, as this lesion represents as a rule only a functional disturbance without any necessary severe anatomical lesion. Lesions of the aortic valves are more liable to develop emboli, lesions of the mitral valve induce lung disease, stenosis of the pulmonary valve entails the danger of tuberculosis.

Concerning individual symptoms, cyanosis is always a bad sign. Cyanosis indicates usually involvement of the right heart, deficient blood supply to the lungs, and defective aeration of the blood. With this defective aeration, changes in metabolism occur at once with accumulation of toxins in the blood. Dyspnœa also indicates a stage of broken compensation. It occurs especially early and severely in lesions which affect the pulmonary circulation, thus especially in mitral lesion. Dyspnœa is less severe in aortic lesions until the later stages. Irregular action and arythmia are not necessarily bad signs, but become bad when the arythmia may not be restored by rest and the use of heart stimulants. An early arhythmia or allorhythmia (pulsus bigeminus, hemisystole), Cheyne-Stokes respiration, are *signa mali ominis*. Tachycardia usually indicates dilatation. Dropsy is by no means of necessity a bad sign, as even an extreme anasarca may be rapidly dissipated when the tone of the heart muscle is restored. But a ruptured compensation can rarely be restored for anything more than a comparatively short time.

In the process of embolism everything depends upon the situation and nature of the embolus, whether simple (bland) or septic. A cerebral lesion may be rapidly fatal or may leave damage which is worse than death. Save in cases of septic endocarditis (puerperium), pulmonary infarctions do not often take life. The hemorrhagic infarction is usually absorbed to leave a pigmented cicatrix, but the

simple embolus may excite an exudative pleurisy, or the septic embolus may cause gangrene or abscess.

The Relation of Pregnancy.—Pregnancy always aggravates organic heart disease. Duroziez called attention to the menorrhagia which occurs in these cases and which may persist even during the course of pregnancy. Sterility is frequent; abortions are more common, sometimes in the very first days, often as late as the seventh month. The frequency of premature birth has been especially remarked by Porak, Duroziez, and Budin. Porak (1880) made observations of the principal events which occurred in 90 cases of heart disease in pregnancy. The condition was stationary 21 times, and was aggravated during pregnancy 63 times, temporarily 46, permanently 59 times. The heart disease was aggravated by labor 12 times, improved during parturition 27 times, very slowly afterwards 9 times. Death occurred before labor 6 times, during labor twice, during the puerperium 25 times. Schlayer showed from the study of 25 cases, and studies also of the cases in the literature, that the dangers of pregnancy and parturition do not depend upon the kind of valve lesion but upon the condition of the heart muscle, that is, upon the degree of compensation. Premature labors occur more frequently in multiparæ than in primiparæ. The same thing is true of deaths. Two patients died in parturition, ten in puerperium, all of whom were multiparæ. Of 17 cases under the observation of Leyden, 16 were lesions of the mitral valve, and of these cases 11 died. Leyden considers mitral stenosis the most dangerous lesion. Death occurs only rarely during pregnancy, more frequently during labor, and most frequently shortly after delivery. The immediate cause of death is either œdema of the lungs or collapse of the heart. When the patient survives she remains feeble for a long time or it is found that the compensation is broken. Pregnancy in these cases justifies, therefore, the induction of premature labor, and the result of this interference is better when it is not postponed to the last moment.

The Relation of Tuberculosis.—The existence of tuberculosis intensifies the symptoms and hastens the progress of valve lesions. The relation of heart disease to tuberculosis has been considered from two extreme standpoints, one as favoring and the other as preventing the development or spread of the disease. The fact is, that heart disease may act in both ways. Any lesion of such character as to interfere with the nutrition of the lungs, as for instance stenosis of the aortic valves, or more especially of the pulmonary valves, distinctly favors the development of tuberculosis. On the other hand, the lesions which produce in the lungs and veins hyperæmia, or stasis, make the soil infertile for the growth of the tubercle bacillus. This fact has been

so often observed in the lungs as to have led to the induction by Bier of artificial hyperæmia in the treatment of tuberculous affections, as of the bones and joints. It might be easy to understand how a defective nutrition would fertilize a soil for the growth of tubercle bacilli. The immunity or apparent immunity of hyperæmic tissue is difficult to explain. Lepine suggests the idea that the bacillus finds it difficult to live in a medium saturated with serous fluid, as the lungs are in a case of heart disease. Bouchard is inclined rather to appeal to the bactericidal properties of the serum of the blood.

Rokitansky had always opposed the view of any antagonism between heart disease and tuberculosis. It is only mitral stenosis that in any way inhibits the development of tuberculosis, while all the lesions which interfere with the blood supply to the lungs, especially pulmonary stenosis, favor it. Wagner (1861) first demonstrated tubercles in the endocardium. Teissier (1895) finds the following forms of tuberculous endocarditis: (1) Tuberculous endocarditis; (2) sclerosis of the endocardium; (3) valve lesions; (4) acute vegetative endocarditis.

Of the first (specific) form there are but thirty-five cases in the literature in which tubercles were actually found, and the bacillus was demonstrated in but six of these. But many of the cases are not fully examined. Teissier could never find the bacillus in the acute endocarditis of phthisis pulmonum, and could not find it in that of miliary tuberculosis. Moreover, he could not produce tubercular endocarditis experimentally. Sclerosis is common especially in the young. It is the effect of a toxin. Endocarditis is, as stated elsewhere, frequent in tuberculosis, but is usually due to secondary infection, especially by the streptococcus, staphylococcus, Bacterium coli commune, and other forms. Hence the valve lesions of tuberculosis do not differ in prognosis—aside from the affection of the lungs—from that produced by other cause.

In all cases the prognosis is determined chiefly by the condition of the heart muscle. Whether this or that valve is affected, whether the lesion be a stenosis or regurgitation, or both, whether there be combined affections, whether the hypertrophy be extreme or slight, in all cases the question turns upon the condition of the muscle of the heart. The aortic valves may be agglutinated to form a funnel whose orifice will permit only the passage of a sound, the mitral orifice may be reduced to a button hole slit, and still offer no great obstacle to the circulation if the musculature of the heart is fairly sound. Muscular structure comparatively sound—sufficiently sound, for instance, to react to digitalis—will furnish a favorable prognosis, at least for a time. Where the heart no longer reacts, the prognosis

becomes bad. The condition of the heart muscle may not be established, with any degree of exactitude, by the nature, extent, or intensity of the valve lesion, but may be alone appreciated by the recovery of the heart under rest and the stimulation of exercise and drugs. The immediate prognosis is determined by the response to digitalis.

The prognosis would depend, therefore, other things being equal, upon the situation of the individual. The patient who belongs to the working classes, and who must force the sick heart to do the work of a sound heart, rarely survives a decade of years. The patient who has used up his heart muscle under the stimulus of alcohol has little chance to survive long. The patient who may adjust his life to his surroundings, who is temperate, philosophical, docile, has the best outlook for a longer life. As in almost all the conditions of life, a single imprudence may undo the good of months and years' construction. Thus Liebermeister narrates a case in which a perfect compensation was destroyed by a single act of folly, against which the patient had been repeatedly warned. This patient in a boast attempted the ascent of a mountain in a certain very short time. The strain was followed immediately by a rupture of compensation which caused speedy heart failure and death. It is said that the celebrated littérateur, Matthew Arnold, precipitated a heart failure in the same way by an act of severe strain in jumping. Unfortunately, the compensation is not always under control. Every practitioner of experience may add to the list similar cases to that reported by Traube of a sudden disturbance of compensation of stenosis of the mitral valve which had lasted for years, in consequence of a violent attack of grippe.

Prophylaxis.

The patient affected with heart disease should recast his life. The wise man adapts himself to the inevitable in his surroundings, and this principle should find application to the interior as well as to the exterior of the body. The patient affected with valve lesion is no longer capable of severe effort. Ordinarily a severe effort does no damage because it makes draft upon the reserve force of the heart. But in disease of the valves the reserve force of the heart is already being drawn upon. The reserve force is reduced or is actually consumed in discharging the ordinary work of the heart and wants of the body. It will be remembered in all cases that the gravity depends upon the condition of the heart muscle. Bouillaud remarked long ago: The location of the precise seat of contraction in one or the other cavity of the heart is a question which is essentially more curious than useful (*qui est au fond plus curieuse qu'utile*). The prophy-

laxis as well as the prognosis must have reference to the myocardium. The general principles of prophylaxis have been laid down in the conservation of hypertrophy and described in the study of this condition.

The subject of valve lesion must preserve the muscle of the heart as much as possible. The workingman or woman has in this regard but little chance. The prophylaxis as well as the prognosis must have reference to the social position. But all is not in favor of the rich, as under the inexorable law of compensation in nature, they run into the other extreme of inactivity which is almost equally dangerous, as luxurious habits favor arteriosclerosis. The patient will often be obliged to change his vocation. In this respect he must avoid the extremes of excessive muscular strain and close confinement to the house, but in a general way the clerical life is better.

While the disease process is active, as in its inception or during periods of exacerbation, the patient must observe absolute rest. He should go to bed. There ought to be no compromise in this matter, but where the patient will not go to bed he should adopt the recumbent or semi-recumbent posture the greater part of the day. Under rest the heart has a chance to regain some reserve force. The whole principle of treatment in this stage of disease is summed under rest, and in the observance of it the main element in prevention is secured.

There are a hundred ways of taking the strain off the heart. Some of them come into consideration every hour of the day. They may begin with the first hour.

The patient may be allowed more sleep. Insomnia exhausts the heart as well as the brain. Sleep is nearly absolute rest and up to certain limits the more sleep the more rest. The patient should sleep in a cool, but not cold, and dry, never damp, room, preferably in the second story of the house, not higher, as the climbing of stairs is hurtful to the heart. He should be lightly but sufficiently clad in a garment which has not been worn during the day, and in a thoroughly well-ventilated room without draught. The room should be inundated with sunlight during the day and should be perfectly dark during the night. The patient should sleep an hour later in the morning. He may begin the day with the restraint of movement. He should lift himself from bed gradually, not suddenly, and should dress slowly, not rapidly. Before dressing he may take a warm, tepid or cool, but not cold, bath. On retiring at night he may take a warmer, perhaps almost a hot bath. The morning bath should be followed immediately by breakfast, which should consist of fruit, oatmeal, a soft boiled egg, and a light roll. A piece of breakfast bacon or small fish may substitute the egg. The breakfast may include a cup of coffee, not too strong, diluted rather with twice or

three times as much milk with cream. After the fast of the night is thus broken the patient should rest half an hour, during which time the bowels should be moved. A natural tendency to constipation, which results from the more sluggish mode of living, may be overcome by a draught of Carlsbad water before breakfast, by a small pill, one-fourth of a grain, of podophyllin at bedtime, or by a gelatin tablet or a few drops of some fluid preparation of cascara sagrada. Calomel is always a good laxative in heart disease but may not be taken continuously; sometimes a half a grain at bedtime will suffice.

Hereupon the patient will betake himself to his avocation, in the practice of which he will keep himself under restraint, physical and psychical. Efforts of restraint are difficult at first, but like everything else, grow easy on exercise and after a while become habitual and then are "second nature." All the emotions affect the heart. Depressing emotions especially degrade the heart, but of the sudden emotions those of elation are the more dangerous. Dinner should be taken at noon and should be a more substantial meal. The dinner should be prefaced by a plate of soup and should further consist of fish, roast beef, or roast lamb, fowl, broiled, boiled, or roasted, nothing fried, and of a few vegetables, potatoes, asparagus, celery, cauliflower, young green corn, tomatoes, with but sparing use of the heavier vegetables, cabbage, beets, carrots, peas, and beans. Preferance should be given to animal food. The drink may be tea, weak, that is, diluted freely with milk, or milk itself, which plays such an important rôle in the treatment of advanced cases of the disease. After dinner the patient should rest half an hour to an hour. A light nap of ten to fifteen minutes' duration may be taken, but anything like a heavy or prolonged sleep should be avoided. Under all circumstances heavy sleeping after eating is forbidden, as such sleep favors fatty degeneration. The consumption of fat is lessened in sleep, as is also the giving off of carbonic acid and the taking up of oxygen (Stofella). At the end of an hour the patient may return to his avocation. He is fortunately situated who is able to return home at an earlier hour, finding time, if necessary, for some light exercise. The supper at six o'clock should be light, a piece of cold chicken, a couple of sardines, a piece of toast, and a cup of diluted tea. The evening may be spent in entertainment. Happy is the individual who may find it at home. The patient should go to bed at ten o'clock, and go to sleep at once. To secure this object it may be remembered that an easy conscience makes a soft pillow; it may be further enjoined that service at the shrine of Venus may be rendered only in response to natural demands. Every practitioner may recall cases of sudden death from "heart failure" at this time.

Treatment.

From the fact that valve lesions are organic and in their nature incurable, it might be assumed that the chapter of treatment would be of necessity unsatisfactory. It is certain that no drug may secure the absorption of a thrombus or a fibrinous deposit. It is still more certain that no drug may dissolve out an atheroma, dissipate a cyanotic induration, or substitute cicatricial with sound tissue. In old times, before the nature of the disease process was known, such an idea was entertained, but pathology displaced it by disclosing irremediable lesions, and this gloomy prognosis contributed to the despair of therapy. The way out came from a consideration of the etiology through the avenue of prophylaxis. It was seen, for instance, that the changes of atheroma, while they could not be prevented altogether, might be postponed or inhibited by the carrying out of the proper treatment of alcoholism, syphilis, Bright's disease, gout, etc. The knowledge of the relation of the infections was a great contribution to therapy, and the tracing of most cases to the action of pyogenic micro-organisms disclosed new lines of attack in therapy as well as prophylaxis. The knowledge of the almost specific virtues of salicylic acid in the cure or control of rheumatism has been of incalculable advantage in the prevention of endocarditis, if only by curtailing the duration of rheumatism. When it shall have been established that rheumatism itself is a cryptogenetic sepsis, the cause of the disease will more frequently be hunted out, discovered, and removed. The pneumonic and tuberculous cases may be more readily determined in the discovery of depots of infection in other parts of the body.

The *indicatio causalis* addresses itself, therefore, to the treatment and relief of rheumatism, pneumonia, tuberculosis, with the various infections of sepsis, some of which are obtrusive or may be disclosed to thorough search (gonorrhœa, prostatitis, salpingitis, bone caries, etc.).

It is not at all improbable that the cause may be directly addressed by means of the serum therapy now so successfully employed in the treatment of diphtheria, tetanus, etc. Mirouoff, Charrin, and Roger and Marmovek have finally succeeded in securing from immunized horses, mules, and asses an anti-streptococcic serum which introduced into the blood in quantities of 50 to 100 c.c. cured or cut short attacks of puerperal fever and erysipelas. It is too soon to make more than mention of these things.

But in chronic valve lesion the damage is done. It is no longer

a question of prevention. Prophylaxis is more a matter of preservation by limitation of extension, more especially by way of prevention of relapse. There is, therefore, no question of meeting the *indicatio causalis*, and therapy resolves itself into the treatment of symptoms with sustentation of the strength of the heart and of the whole body, of which the heart remains, no matter what may be said to the contrary, the chief organ.

The knowledge of the fact that the future of a case depends primarily upon the condition of the heart muscle and only secondarily upon the character, situation, and extent of the valve lesion, concentrates the endeavors of therapy.

The first maxim of therapy, which was inculcated by Hippocrates, has special reference to the treatment of diseases of the heart, namely, *first, do no harm.* So long as the muscle of the heart compensates the lesion it should be religiously let alone. Nowhere else may interference be so meddlesome and mischievous. Because a man has heart disease it is not necessary to treat it. It is everywhere a safe rule in therapy, when there is no specific, to treat the patient and not the disease. Sometimes the only thing to treat in a case of heart disease is the fear and apprehension which has been created by a knowledge of the extent of the disease.

Nevertheless, knowledge is power. The patient should be acquainted with the fact that the heart is affected, but in the right way. He may be informed at once that the old idea of the necessarily fatal nature of valve lesions has been entirely dispelled and that the subjects of heart disease often live to advanced life. As has been seen in the discussion of prophylaxis, the patient holds his fate in his own hands. The chief treatment requires a readjustment of the modes of life to the altered conditions. Enough has been said upon this subject elsewhere. No general rule may be laid down. Unfortunate is the patient who falls into the hands of a practitioner who prescribes digitalis because the patient has heart disease; happier is he who consults a physician who is conscientious and who will study his case. The substance of this comment may be summed in the statement that sometimes the best treatment is no treatment at all.

But while it may be said that there is no class of cases in which so much damage may be done by ignorant interference, it must also be admitted that there is no class of cases which may be so much benefited by intelligent direction. It is true that certain cases are curable. Infected valves may be stretched or agglutinations may occur which make up for defects in the valves. Thrombotic deposits may be discharged into the circulation and leave the valves clear with but little or with reparable damage at the seat of deposit. But these

cases represent the great exception. They are individual and may not be included in the general estimate, so that treatment is directed to meet symptomatic indications.

So long as the valve lesion is not compensated, absolute rest must be enjoined, and any attempt at exercise by gymnastics, with the view of furthering hypertrophy, is forbidden. Broken compensations, again, demand absolute rest and they may be restored alone in this way. Absolute rest means rest in bed. The one essential, *sine quâ non*, in the treatment of rupture of compensation is rest in bed. Rest alone suffices to restore most cases. Under it the quantity of urine increases, the dyspnœa ceases, the hydrops disappears. In one case under the observation of Liebermeister the patient insisted upon leaving his bed prematurely, in the course of a few weeks, and returning to his office, whereupon he fell down and broke his leg on the steps, and this accident put him back in bed for a long time. The accident was, however, a blessing in disguise. The patient recovered and was, at the time of the report, engaged in his office, fifteen years after the first disturbance.

In this connection may be quoted, as illustrative of the value of rest alone, the statements and statistics of Roemer, who makes the following report of 230 cases of ruptured compensation in valve lesion treated in the clinic at Tübingen (1870–1890 inclusive). Of these cases 130 were treated by the expectant method with rest in bed and suitable diet, without the use of any active drug, with the result that compensation was entirely restored and sustained for a long time in 80 of these cases; the effect was only temporary in 23 cases, and in 27 cases satisfactory results were obtained only after the use of drugs (Liebermeister). Pospischil, too, speaks of cases in which the hydriatic and mechanical treatment accomplishes results in desperate cases where all the other remedies of materia medica have completely failed. The application of cold, the use of gymnastics, massage, and hydrotherapy, with milk diet, suffice to dissipate entirely the signs of relative insufficiency of the tricuspid valve.

In all cases of chronic affection the cardinal factor is the support of the heart muscle, and in such a way as to avoid exhaustion. Thus the general principle of therapy should be that which is understood by a tonic rather than by a stimulating treatment.

New tone is imparted to the heart in two ways: first, by reduction of work; second, by support of the heart muscle. The reduction of work is effected in the readjustment of life. This question has been studied already in prophylaxis, but is so important as to justify detailed mention even at the risk of some repetition.

The subject of heart disease is in a sense a cripple. The patient

affected with heart disease will spare himself at least all superfluous effort. He will keep himself under restraint by voluntary effort until, as stated, the restraint becomes a habit. Thus, he will not rise suddenly from the recumbent or sitting posture; he will be more deliberate in all his movements; he will not climb stairs more than is necessary, nor mountains nor hills, except under supervision; he will indulge in no gymnastics, except as directed in treatment; he will not ride nor swim nor dance immoderately; he will take systematic exercise and make it as pleasureable as possible, but he will avoid fatigue; he will avoid lifting and stooping and straining; he will rest after meals; he will abstain from all heart stimulants, alcohol, tobacco, coffee, tea; he will be especially careful regarding sexual intercourse—*omne animal post coïtum triste est; coïtus prolongatus et reservatus* are especially enjoined; he will be a man of exemplary habits and have every passion well under heel. He will know that present enjoyment at the cost of future suffering should certainly be avoided. The subject of heart disease will especially stand guard over his emotions. All sudden emotions excite the heart. Depressing emotions exhaust the heart. Men, more frequently women, do die of grief. Strange to say, of the sudden emotions, those of joy and exaltation are the most dangerous to the heart. At the same time it will be remembered that pleasure is the best tonic. Cheerful surroundings, congenial associations, the gentle intellectual stimulus of conversation, entertaining and instructive reading, good music, excite in many reflex ways nervous impulses which support the heart and evoke from it the gentler efforts which secure increased blood supply to the brain.

The clothing should suit the season. The patient should be at all times protected against exposure by light underwear, even in summer. Underwear is as a rule better than overwear, but too heavy underwear out of season excites profuse sweat, exhausts the patient, and increases the tendency to take cold. A fresh gown next the skin at night has the virtue of a bath.

Bathing stimulates the skin and through the skin the heart. The bath should be warm or tepid and should be taken preferably at night, that it may be followed by a gentle and safe diaphoresis. The cold bath may be permitted in only the most robust cases. Hot baths have special value in the treatment of advanced stages of the disease.

The heart is spared work by change of climate, especially to avoid the rigors of winter and vicissitudes of fall. A soft and balmy air is the best climate for valve lesions. Moderate elevations may be advised in mild cases, but mountain air is too stimulating

for advanced cases. Mountain air excites the nervous system and produces palpitation with precordial distress. To this condition, however, tolerance may be got in time. For the milder cases the air of valleys is the best, especially of valleys with lighter elevations convenient for short climbing, with here and there a piece of woods, with alternate shade and sunshine and with occasional resting-places. Certain watering-places furnish these indications, as in Europe, Kissingen, Ischl, more especially Nauheim and Oeynhausen, which have the advantage also of hot baths rich in carbonic acid gas. These indications are met also, in our own country, at the hot and warm springs of Arkansas and Virginia. Many patients are most benefited by the more southerly seaside resorts, Cape May, Virginia Beach, etc. Advanced cases are more improved by a stay of some duration in the islands of the sea, the Bahamas, the Bermudas, the Canary Islands, the Azores, etc. The valleys and tablelands of Southern California furnish varieties which may be adapted to an individual case. Patients whose means permit may change residence winter and summer, and may find every climate and condition in our country, from Bar Harbor in Maine to the southern points of Florida and California.

The diet should be simple, plain, and nutritious. Milk fulfils every indication. An exclusive milk diet is especially indicated in cases complicated with disease of the kidneys. Many patients are rescued from conditions of great danger and brought into absolute safety by an exclusive milk diet. The patient may in these cases take two or three quarts of milk per day. The Russian physicians, Karell and Högstedt, especially recommend the absolute milk diet for its diuretic effect in the relief of dropsy. Koppel gave the Karell milk diet in a case of myocarditis which he succeeded in relieving after failure with every other treatment, including rest in bed. Koppel recommends at the start 600 to 800 grams (20 to 26 ounces) of milk per day. This quantity is rapidly increased to 1,500 grams, and is continued regularly; but there is given also 100 grams (3 ounces) of zwieback (toast) to cover the deficit in carbohydrates. Buttermilk is a good substitute in summer time, and kumyss, kephir, fermented preparations, or wine whey may fulfil the indications when the necessity for light stimulation has been reached. As a general rule, the diet should consist chiefly of animal food, with preference for the white meat of fowl and fish. Heavy meats and fats should be taken sparingly. Vegetables and fruits should be selected with care. Stewed fruits may be allowed when fresh fruits are forbidden. Light custards may be eaten, but pastry should be put under ban. The bread should be white and light. Brown bread

or the heavy vegetables, cabbage, carrots, turnips, beans, anything which has a tendency to produce gas, should be avoided. The supper should be very light and should be taken several hours before retiring.

Hirschfeld found that change of food with limitation in quantity was followed by the best results in the treatment of heart disease, as patients who took only small quantities of scraped meat, soup, and eggs, soon showed increase in the quantity of urine. But the change of diet was without influence if there was no œdema, and heart weakness was manifest only by dyspnœa. Hirschfeld holds that the raising of the blood pressure and *luxus-consumption* lead to hypertrophy through plethora. The scientific foundation for a reduced nutrition follows thus of itself. Under all circumstances, a reducing treatment should be used with great caution. Glax insists upon it that the diminution in fluids is one of the most essential features in the treatment of chronic heart affections, and this method alone may suffice to secure compensation, even in cases in which the ordinary heart stimulants have lost their power. In the adoption of this method the drinks must be regulated to the excretions. It is wise in using these means to weigh both ingesta and egesta.

Such extreme precautions are as a rule impracticable, but patients themselves may learn to practise them when impressed with the fact that they may by such attention for a short time prolong life for years.

The best drink is water (*ἄριστον μὲν ὕδωρ*), but the water should be pure—that is, uncontaminated by animal or vegetable matter, but properly impregnated with the natural salts. The water may be taken cold, and should always be cool. Cold water is a gentle stimulant to the nervous system. Ice water is not injurious if taken slowly; it becomes warm before it reaches the stomach. But the sudden inundation of the stomach with large quantities of ice water is harmful. The drinks in summer may be acidulated or carbonated. Cool acid drinks favor digestion and facilitate peristalsis. Acid drinks are all the more indicated in summer when the food is, or should be, largely vegetable. Acids help to hold the phosphates in solution. Carbonated drinks in small quantity may be recommended. Carbonic-acid gas gives to water its sparkle and sapidity. It has also remarkable solvent properties. It will disintegrate flint by displacing silicic acid. Hence carbonated waters actually assist the digestion of vegetable food; besides, they stimulate peristalsis. Of course they should never be taken in heart disease in such quantities as to distend the stomach with gas. Acid drinks are represented in lemonade, in acid phosphates, in soda water with certain fruit syrups.

Carbonated drinks are supplied in soda water, Vichy and Apollinaris waters, etc. In all cases, as stated, the quantity of fluid should be lessened, as distention of the blood-vessels subjects the heart to extra work. It is not advisable, however, to punish the patient with the thirst cure, as recommended at one time.

Later in the treatment of heart disease, when the action of the heart begins to flag, that is, in the stage of compensation, the food must be more nutritious and the drinks may be made more stimulating. In the absence of any affection of the kidneys, eggs may be allowed, with beef, mutton, and the stronger meats, and small quantities of alcohol may be administered in the form of pure beer or the lighter wines, Catawba, claret, Sauterne, the Rhine wines, and so on, up through Madeira, sherry, Tokay, to whiskey and cognac, as the degree of exhaustion may demand. Especial reference is had to the failing heart when it is said that "wine is the milk of old age."

Attention must be paid to the bowels, which should be evacuated at least once every day, and in the case of stasis or dropsy preferably twice. The natural tendency to constipation, the result of the inactivity of the body, may be overcome by change in the food, as by the use of oatmeal, stewed prunes, apples, etc. Sometimes the grape cure, that is, the ingestion of large quantities of grapes in the right season, effects a pleasant cure of this condition. More obstinate cases may be effectually relieved by the injection of one pint of *pure* olive oil, which is allowed to flow in slowly from a fountain syringe, the patient lying first upon the left side, later upon the back, and then upon the right side, that the oil may traverse the whole tract of the large intestine. The injection should be taken on retiring and the oil should be retained all night, a matter of some slight difficulty at first, but later of easy practice on account of tolerance. These injections are usually followed by the evacuation of a large golden-brown stool on the following morning.

Legion is the number of drugs which may furnish relief. Certain patients are most benefited by the use of mineral waters, Carlsbad water or salts, Friedrichshall, Hunyadi János, etc. Other patients prefer vegetable drugs, of which eligible preparations are found in the extract of cascara sagrada, rhubarb, aloes, podophyllin, the compound licorice powder, the virtue of which depends upon senna. Calomel is always a good purgative in heart disease. It has also a diuretic action.

The heart muscle is best toned by baths and by exercise, and the finest results are secured in institutions where they may be obtained together and applied and adapted under intelligent supervision.

The *balneotherapy* of heart disease began with the statement of

Beneke (1859), who declared that he would not hesitate a moment to send patients affected with heart disease in consequence of rheumatism to baths. Beneke published his first work upon this subject in 1870, and thereupon large numbers of patients visited Nauheim, so that he was able to make a report of one hundred and one cases as early as 1872. The next works were by Groedel and Schott, who distinguished the Nauheim bath cure as a tonic of the first order for the weak heart. From the numerous observations made in Nauheim it was shown that the carbonic-acid thermal baths soothe and regulate the action of the heart and decidedly invigorate the cardiac muscle. The idea is that in the beginning of the bath the vessels of the skin contract and thus increase the intracardial pressure, but that the work of the heart is lightened because the skin is secondarily rendered hyperæmic under the irritation of the carbonic-acid gas. This mode of gymnastics for the heart is adapted to all cases of disturbance of circulation which depend upon incompetency of the heart muscle. It is a matter of indifference whether the incompetency depends upon valve lesion, muscle disease, or is the result of general disturbance of nutrition or disease of the vessels (Pollatschek).

Jacob suggests that the carbonic-acid bath does not act only upon the circulatory apparatus, but that in addition to its influence in dilating the cutaneous vessels it exercises also an indirect influence on the vagus and accelerator nerves. These baths distinctly diminish the frequency of the pulse, increase the length of the diastole, elevate the pulse pressure, and enlarge the pulse wave.

Where it is impracticable to visit the baths the system may be used at home. Lippert and Sandow advise artificial carbonic-acid baths.

Great caution must be used in the beginning of this treatment. The bath should contain but a small quantity of salt and carbonic acid and the temperature should not be above 32° to 33° C. (about 90° F.); moreover, the bath should not last longer than thirty minutes. Patients with excitable hearts are always much benefited with lukewarm baths, 22° to 32° C. (71° to 90° F.), of short duration. Simon absolutely rejects sea baths and warm baths in the treatment of organic heart disease of children.

But even the hot bath may be indicated in certain cases. It is certain that heat properly applied is one of the most effective remedies in stimulating and securing tone to the heart. Heitler especially deserves credit for having demonstrated the virtue of hot applications and hot baths in the treatment of valve lesions, atheromatosis, myocarditis, and Bright's disease. The value of hot baths in

Bright's disease has always been known, and the frequency of association of hypertrophy of the heart and myocarditis with affections of the kidney might have sooner suggested the use of hot baths in these affections. Heitler finds that hot baths of the temperature 32°–38° R. (102°–112° F.) of forty minutes' maximum duration, distinctly increase the tone of the heart muscle. This increase of tone is indicated by the diminution in the dulness of the heart, which is not temporary only, but remains permanent.

Sometimes the increase of tone may be recognized by percussion for three-fourths of an hour. Under this increase the arhythmia and tachycardia disappear, the pulse becomes full and strong. It is true that it becomes weak again after cessation of the baths, but it is nevertheless better than before. Thermic stimulation may be used also in the form of hot applications, but hot applications in the region of the heart have only a very temporary effect unless the precordial region be at the same time subjected to friction with cloths.

Exercise in the treatment of heart disease was first recommended by Stokes (1851), who suggested the practice of regular gymnastics and made the first mention of the good results which followed journeys on foot in mountainous lands during which he said the patient "got his second wind." Cohnheim next (1866) called attention to the fact that the heart, like every other muscle of the body, must grow with work. For every strong muscular action must increase the action of the heart, he says, and in frequent repetition lead to increase in volume. Hereupon Franz and Schott (1881) remarked the increase of tone and lessening of frequency of the pulse under "quiet or tranquil exercise," and Oertel emphasized the value of climbing mountains. Franz tells the story of a lady from Holland who was sent to the mountains with instruction from her physician never to take a step in exercise but always to sit or lie about in the open air. This patient suffered from extreme dyspnœa on the slightest exercise and finally fell into melancholy seeing that nothing could be done for her. She was now instructed to take daily a few deep long breaths, and the effect of this suggestion became soon apparent in allaying the dyspnœa. The habitation was now changed to a point which necessitated a little climbing with the effect of tranquillizing and strengthening of the pulse almost at once. The retardation in the action of the heart wherein time is gained for full diastole is the best criterion of the character and gauge of the amount of exercise. But the character and amount of the exercise should be supervised at first by the physician and should be graduated to the condition of the heart. The best gymnastics are found in the so-called apparatus of resistance, in which the muscle work may be measured. The

different effects of different forms of gymnastics are also referred to an excitement of the vagus from the sensitive nerve fibres in the muscle. Outdoor exercise secures the benefit also of extra oxygenation of the blood and increased metabolism. Thus when hypertrophy has occurred, there may be at first passive and later active motions, but forced exercise is always dangerous. Oertel's training method gives at times almost incredible results, but in some cases too much caution cannot be exercised to avoid mishaps. Patients are not to be driven to exercise in the face of palpitation and dyspnœa. Schott says that the injurious influence of the Oertel cure was established in eight of his cases and Liebermeister declares that many patients with heart disease have been literally driven to death in the last ten years.

A variety of massage in the form of continued tapping or percussion, is another means of increasing the tone of the heart in early or in aggravated stages of insufficiency. It was observed in a series of experiments that the absolute and relative dulness both diminish sensibly within two months under tapping of the precordial region. A permanent result was observed in one case of severe myocarditis which had not been favorably influenced by hot baths. The plan is on the principle of the mechanical gymnastics, which may be applied in various ways in the treatment of heart disease. As mitral lesions are especially distinguished by stasis in the lungs, Campbell finds that he is able to further the circulation in the lungs by regular, deep inspirations. Methodical efforts in breathing, singing and speaking assist in this method. Obesity is a hindrance, as is also tight lacing.

Gentle faradization of the surface is another method of stimulating the heart. v. Bandler found in his studies of the heart of the daphne (water flea) whose action is visible on account of the transparence of the body of the animal, that ether, chloroform, chloral, alcohol, amyl nitrite, helleborein, nicotine, atropine, and muscarine were poisons to the heart upon which they acted exactly as in the case of the vertebrates. The interrupted current of electricity relaxed the heart while the induction current produced contraction and increased the frequency of the action (Dreser).

In the treatment of symptoms, palpitation is quickest controlled by cold, especially by the application of the ice-bag or Leiter coil. Where the attacks are frequent the patient may secure relief by wearing a hollow tin shield filled with cold water. A flask of ice water is a convenient substitute. When the nervous element predominates, relief may be secured by the bromides. It is, however, not good practice to continue the use of any remedy which may

affect the stomach. The attacks of palpitation with the other signs of irritation or exhaustion disappear under the radical treatment of the condition just described.

Pain which varies in every degree of character, from the vague distress which is known as precordial anxiety to the excruciating agony of angina pectoris, which may literally transfix the patient, is relieved in its lighter forms by counter-irritation, as by the application of a mustard paste, or if more continuous by the application of a belladonna plaster, and in its severer form by the use of morphine internally or subcutaneously, and more happily by the nitrites, especially the amyl nitrite, gtt. ii.–v. of which are inhaled from a pocket handkerchief. Recurrent attacks of this character may be prevented by the use of nitroglycerin, gtt. i.–iii.–v. of a one-per-cent. solution three or four times a day. A convenient form but one not quite so reliable is offered in the tablet or pastile, 0.001 each. The systematic treatment in this way calls for an administration of one a day for five days, then two a day for five days, and so on up to four per day, whereupon the dose is diminished one tablet every five days.

Attacks of tachycardia may often be controlled in the same way by the application of cold, by a mustard paste, or by some quick stimulant, a glass of cognac, Hoffman's anodyne, gtt. xv.–xxx. or compound spirit of ether, most pleasantly in sugar water. Faradization of the pneumogastric nerve in the neck will sometimes cut an attack short. The attacks of tachycardia are also wont to subside under the proper toning of the heart.

Bradycardia, which is rare, may be addressed also with diffusible cardiac stimulants, especially with caffeine, and is sometimes relieved by the straining efforts described under the neuroses. Mild cases yield to phenacetin and camphor with warmth and counter-irritation. Care will be taken in these cases to avoid any sudden abrupt change of posture, especially from the recumbent to the upright, which has not infrequently been followed by almost instantaneous death. Too rapid action of the heart may necessitate the use of morphine; too slow action of atropine to allay irritation of the vagus from constriction at the aortic orifice (Rummo).

Dyspnœa is controlled by exposure to fresh air, especially by absolute rest and by the use of the cardiac stimulants, alcohol, ether, musk, camphor, caffeine, etc. The quickest effects may be obtained by subcutaneous injection, but great care must be exercised in the use of ether in this way, as it sometimes irritates intensely and not infrequently induces necrosis of tissue. In all cases the ether should be injected deep. Where the effects are not produced at once, repeated injections should not be made, as in large dose ether paralyzes

the heart. As to musk it is almost impossible to get a pure tincture free from the dangers of sepsis so that resort is best had to camphor and caffeine.

Camphor may be given in the form of the simple tincture ʒ i.–ʒ iv. largely diluted with water, or may be injected subcutaneously with pure olive oil in the form of the oleum camphoratum. Přibram declares that the injection of camphor oil in dose of 1 cm. and over is never followed by the least bad effect, and that he has seen it absorbed when given on the day of death even up to two hours before the end.

One of the best remedies in the treatment of cardiac dyspnœa is quinine, either alone or in connection with caffeine or codeine muriate. Should these remedies fail, resort may be had to morphine subcutaneously. Light chloroform narcosis practised with caution sometimes furnishes excellent service. Most remarkable relief is often given in many cases by the application of cold compresses to the chest. Sturges found relief in children of the dyspnœa which occurs in attacks and sometimes ends fatally, by the use of leeches, while the application of cold was of dubious effect. Sometimes the subcutaneous injection daily for a long time of a few ounces of a six-tenths of one per cent. solution of common salt anywhere over the abdominal surface, furnishes excellent results in protracted cases.

Huchard describes a form of dyspnœa produced by toxins from defective action of the kidneys. In these cases, all foods which contain these toxins should be excluded. The poisons of the intestinal canal should be neutralized by antiseptics and the secretion of the kidney should be stimulated to eliminate the toxins. A milk diet should be used in the quantity of three to four pints a day. The patient may drink six ounces of milk every two hours in small quantities at a time. With the subsidence of the dyspnœa in the course of ten days a special diet is given of vegetable soup or purée, a few fresh, soft-boiled eggs, and fresh meat. Wild fowl, cheese, and preserves are excluded. The milk diet should be used for five or six days in every month to secure diuresis and the elimination of the toxins. An intestinal antisepsis may be secured by salol gr. v. or hydronaphthol gr. v. every two hours. It will be remembered that hydrochloric acid in small doses, gtt. ii.–x. after meals, is not only an eupeptic but is also a fine antiseptic.

The worst dyspnœa is that due to œdema of the lungs, which may set in at any time in paroxysms of cardiac asthma and usually does occur as a most distressing picture toward the close, *sub finem vitæ*. This dyspnœa calls for the stronger stimulants, especially for cognac and caffeine. A cup of black coffee containing a tea- to a tablespoon-

ful of cognac may secure the action of both stimulants. But caffeine, the active principle, is more powerful. Of the principal coffees the native Ceylon contains caffeine 0.87 per cent., Java 0.25 per cent., yellow Java 0.7 to 0.8 per cent., Mocha 0.2 to 0.6 per cent. Pasta guarana contains as much as 5 per cent.

Stuhlmann and Falck found that caffeine in large dose increased the frequency of the pulse in health, and raised the pressure of the blood by its direct effect upon the heart muscle, independent of the vagus.

Caffeine also distinctly raises the temperature from one to two degrees, and for this reason black coffee has long been recommended as an antidote in poisoning by alcohol, chloral, opium, or other drug which depresses the temperature. Caffeine is especially indicated in the treatment of heart disease where defective secretion of urine may be referred to inactivity of the heart. Riegel in his extensive investigations found that the indications for the use of caffeine were about the same as those for digitalis. The chief difference lies in the fact that caffeine acts much more quickly and is free from any cumulative effect. It was seen that caffeine sometimes helped when digitalis failed. Becher and Lepine confirmed this observation. Caffeine acts best in repeated small doses. But it may be given in much larger quantity than hitherto employed. The sodium benzoate is the form usually preferred, but the cinnamate and salicylate have their advocates. Becher gave the so-called citrate in doses of seven grains and as much as thirty to forty grains per day. An improvised preparation of the double salt, the sodium benzoate, may be made as follows:

℞ Caffeinæ purissimæ, ... 0.5
Sodii benzoatis, ... 1.0
Aquæ destillatæ, ... 150.0
Syrupi simplicis, ... 10.0
M. S. Tablespoonful every hour.

Unfortunately, though the objection is not serious, caffeine produces a contraction of the arteries which leads to venous plethora of the abdomen. Aubert had long ago called attention to this factor as favoring the development of hemorrhoids.

Rummo made a thorough experimental study of the combinations of caffeine with iodine and found that iodocaffeine exercises its chief influence on the nerves, iodotheobromine the muscular part of the heart, while iodotheine uniformly affects both. Iodocaffeine is therefore indicated when the desire is to reach a full diastole, as in insufficiency of the mitral valve; theobromine is indicated when the desire is to strengthen the systole, as in insufficiency of the aortic

valves. As a side effect has been observed spasmodic cough, which is attributed to the secretion of the iodine by the mucous membrane of the respiratory organs.

Among other analogous preparations a first place must be given to the sodium salicylate theobromine known as diuretin, a remedy which became deservedly popular almost at once. Diuretin is advised especially in cases of heart weakness marked by arhythmia and dropsy when digitalis has been used without effect or is contra-indicated from any cause. This remedy sometimes shows surprising results in these cases as it affects the heart muscle itself, so that dropsy often disappears under it entirely in the course of a few days. Thus in one desperate case in which digitalis, camphor, and caffeine availed nothing, diuretin brought about a relative recovery. The diuretic effect is observed between the second and sixth days. Anything like unpleasant effects, nausea, vomiting, and light excitement, are rarely seen, and when they occur usually disappear spontaneously under the continuous use of the drug (Pawinsky).

Diuretin is best given in the form of the powder in the dose of gr. xv. every four hours in soda or seltzer water discharged from a siphon bottle.

Bronchitis, catarrhal affections of the lungs, etc., are addressed with the preparations of ipecac, ammonia, and squills. Ipecac is a light and safe expectorant which may be given in the form of the tincture or syrup, gtt. xv.–xxx. every two or three hours. Where there is more continuous irritation the ipecac may be given in the form of Dover's powder gr. ii.–iii. at a dose; or the syrup may be combined with the syrup of Dover's powder, which is now prepared in the proportion of gr. v. of the powder to ʒ i. of simple syrup. Apomorphine in the dose of one-twelfth to one-fourth of a grain may be given every three or four hours. Apomorphine has a soothing but nevertheless a rather depressing effect upon the heart. Senega is a stimulating expectorant and ammonia is still stronger. The carbonate of ammonia is given in the dose of gr. v. in milk every two hours; but a far more eligible preparation is the liquor ammonii anisati of the German Pharmacopœia, which may be given in doses of gtt. ii.–iii. in sweetened water every hour or two, or every quarter of an hour to bridge over an impending collapse.

Squill acts upon the heart like digitalis, but it has also, according to Heinz, a direct irritant action upon the cells of the kidney, producing in large dose nephritis and hæmaturia. Hence it is contra-indicated in acute or subacute disease of the kidney. Besides its influence as an expectorant squill acts as a diuretic and was used for this effect in antiquity. In the dropsy of uncompensated heart

failure the drug is ineffectual, but in compensation it renders good service with digitalis (Heinz). Squill is also contra-indicated in irritable states of the stomach and intestine, diarrhœa, etc., hence it has been pretty much displaced from the modern materia medica by better drugs. It is usually prescribed in tincture. Sometimes it is combined with ipecac as in the compond syrup of ipecac. It may be given to children in the form of the oxymel, one part acetum scillæ, two parts purified honey.

Where the kidneys are sluggish (Bright's disease) or in the presence of oliguria from stasis, with the dangers of uræmia, the best remedy is nitroglycerin, trinitrin, in the dose of gtt. i.–ii. or as many as to gtt. iii.–v. diluted with water or alcohol every two to four hours. The extraordinary virtue of the milk diet must be borne in mind in these cases.

Dropsy which has proved defiant to rest and the milk diet generally disappears under the use of the heart tonics, diuretin, caffeine, and digitalis. In certain cases no drug is as good as calomel, which acts as a diuretic probably by neutralizing the toxins which interfere with secretion in the kidneys. Calomel is given as a diuretic in the dose of gr. iii. three times a day, and the effect is asserted at the seventh to the ninth dose. Any undue irritant effect upon the bowels may be restrained by opium, and ptyalism may be avoided by the use of chlorate of potash, by frequent cleansing of the mouth, and by touching any ulcer that may form with (caustic) chromic acid. The calomel should be discontinued after the effects are secured. If the dropsy should still prove refractory and threaten to break the skin or interfere with the action of the diaphragm, etc., it should be attacked mechanically.

Unanimity as to the best method of mechanical treatment is still lacking. Southey devised fine silver cannulæ for the purpose of drainage. Gerhardt prefers incisions into the subcutaneous connective tissue as more effective than the introduction of small trocars. With the necessary cleanliness these incisions are entirely free of danger. Michael, who advised the trocar, considers as the best means that which discharges the most fluid in the smallest time through the fewest possible openings. Ewald uses the thickest trocars, such as are employed in puncture of the pleural sac. The penetration is not more painful than that from a heavy hypodermic needle. The skin is to be cleansed with ether, alcohol, and sublimate, and the trocar introduced obliquely into the œdematous tissue. The stylet is then withdrawn, and the region about the cannula pencilled with iodoform collodion. Though the tubes have often been allowed to remain for days Ewald has never seen inflammation or

erysipelas. A rubber tube is connected with the cannula to conduct the fluid over the side of the bed. The cannula should be open at the end and not at the side, as cannulæ with side openings are drawn sideways and loosened by the rubber tube. Sometimes it is wise to lay the tube along the leg a short distance and fix it with a piece of sticking plaster before it is allowed to depend from the bed. In this way the cannula remains fixed and all traction upon it is avoided. The oozing after withdrawing the tube, which is sometimes persistent for days notwithstanding the use of iodoform bandaging, etc., and which macerates the skin, may be prevented by inserting a needle across the opening and winding a thread about it in the form of the figure 8. When the cannula is plugged the stylet may be reinserted or the tube may be withdrawn and inserted anew. One or two tubes in each leg or one in the thigh and one in the leg usually suffice to do the work. As much as a litre or more may escape in a few days, and the relief is correspondingly great, as not only is the fluid of the subcutaneous tissue discharged, but remote regions, the abdominal walls, testicles, etc., are thoroughly drained. In one case of grave nephritis several litres of fluid were withdrawn every four weeks for six months, with the effect of finally subduing the threatening symptoms. Rumpf gets permanent drainage by attaching to the end of the rubber tube a funnel which is inserted in a basin of water on the floor. In this way the force of aspiration is secured (Pollatschek).

Although the process of escape is one of slow oozing, the quantity which is discharged during the day is sometimes almost incredible. Successive layers of cloths are soaked in the course of time, and the bed or the floor at the feet of the patient is saturated with serum. The attendants declare that it seems impossible to keep the surface dry. The escape should be slow, for if the œdema be allowed to subside too rapidly, hebetude with delirium and wandering speech may set in for a short time. The quantity that may be discharged and the length of life that may be secured in this way is illustrated in a case reported by L. A. Sayre of general anasarca from hypertrophy of the heart with general atheroma of the arteries, chronic nephritis, and ascites from compression of the liver and portal vein by proliferating peritonitis. Tapping one hundred and eighty-seven times during six years and five months discharged altogether $1,203\frac{5}{16}$ pounds, that is more than six times the entire weight of the patient.

In extreme cases of œdema of the lungs or brain where death seems imminent from suffocation or coma, it may be justifiable to let blood. In old times blood was let in the quantity of several pints in twenty-four hours in the relief of these conditions, and there is no doubt of

the value of venesection in these desperate cases. In the clinic of Liebermeister it was found that in certain cases of insufficiency and stenosis of the mitral valve, when the pulse could no longer be felt and the blood only escaped from the vein in the arm drop by drop, the discharge of 240 c.c. from both arms rendered the pulse palpable and saved the patient. Under the use now of digitalis, the hydrops, the cyanosis, and the dyspnœa disappeared, so that in the course of two months compensation was again restored.

For the relief of anæmia nothing takes the place of rest. It is observed that in anæmia from twenty-five to one hundred per cent. more blood is discharged into the tissues in the recumbent than in the sitting posture. The next best thing is the manufacture of blood, for which nothing is better than a good iron therapy, whereby the Blaud pill may be recommended specially. If these pills cannot be well taken, there is wide field for selection. A fine preparation for a delicate stomach is found in the pyrophosphate of iron and soda. But iron should not be substituted by arsenic or its preparations. In fact, arsenic should not be used at all in heart disease, especially in fatty degeneration, as it may increase the trouble (Stofella). Alcohol, phosphorus, arsenic, distinctly lessen the resistance of the heart wall and produce dilatation.

Chlorotic women should desist from all work, and this rule should be rigid in all cases during pregnancy and the puerperium. Schlayer recommends rest during pregnancy, and, if there is albuminuria, a milk diet. In the presence of heart failure premature labor should be induced unless it follows spontaneously, an occurrence which is rather the rule under these circumstances. Labor is to be hastened, if necessary by the forceps, but the delivery of the child must be gradual. The sand bag should be laid upon the abdomen and should make pressure so as to occupy the space left by the escape of the fœtus, in order to prevent, under the rapid descent of the diaphragm, a too sudden aspiration of large quantities of blood from the thorax into the right heart. Sometimes it may be necessary to make a venesection. Edge advises the induction of abortion as early as the third month. Delivery should then be as rapid as possible, but hemorrhage should not be stopped at once.

Digitalis.—We come now to digitalis, the study of which has been purposely postponed to the last on the principle of the reservation of the best. Without wasting time in the search for superlatives, it may be said that digitalis is the chief weapon in the hands of the physician in combating heart disease. Fothergill, who made the first scientific studies, quotes from Pereira the fact that the foxglove is mentioned in a manuscript written before the Norman conquest, and states

that Fuchsius (1542) first gave it the name "digit-alis" from the German word *fingerhut*, thimble, on account of the shape of its blossom. Withering, of Birmingham, had observed the kindly clinical effects of digitalis as a diuretic when "the anasarcous limbs pit under pressure" as long ago as 1785. Fothergill made his experiments nearly a century later (1870) "only of an elementary character," he says deprecatingly, on birds, fishes, and frogs to disprove the fallacious idea that the drug was a sedative, "the opium of the heart" as it had erroneously come to be considered. Traube saw that digitalis increased the pressure of blood in the arteries, Fothergill found that it contracted the arterioles, and Foster showed that it slowed the pulse by lengthening the diastole. Thus was established the action as a heart tonic. Digitalis is now "the foremost of the cardiac tonics" (Balfour), and more thorough acquaintance with it has only served to fortify its position. It is indeed questionable if any remedy in the whole range of the materia medica gives the physician who makes judicious use of it more perfect satisfaction, at least for a time. But the injudicious use which came of extravagant claims was attended with extreme reaction. Walshe, for instance, in his work on Diseases of the Heart, published as late as 1851, denounces digitalis. "I have not spoken of digitalis," he says. "The action of this medicine, when it really does slacken the circulation materially, is rarely demonstrably beneficial, sometimes seriously mischievous, always hazardous." This wholesale condemnation was based upon the fear that retardation in the action of the heart would favor coagulation of blood in the organ.

We know now, however, that the process of thrombosis requires more than a mere retardation of the circulation, and have, moreover, become familiar with the fact that the heart is not only retarded but is also strengthened under the action of digitalis.

According to Schultz digitalis directly excites the vagus and in exciting the vagus restores the action of the heart. Digitalis affects the nerve both in its centre and at its periphery. Proof is furnished by the fact that if the regulatory system of the heart be paralyzed by atropine, and digitalis be then injected, the retardation of the heart's action is not observed.

With the reduction in the number of contractions the energy of each contraction is increased, that is, the systole is strengthened, and the pulse is affected correspondingly. In fact, the pulse may be reduced by digitalis as low as to 40 beats (Balfour), to 37 beats (Mavré), to 28 beats (Hutchison), or in extreme cases even as low as to 17 beats (Piedaquel and Heurteloupe).

As to the secretion of urine, it may be said that a single large

dose or small repeated dose has no particular influence. But Brunton found that when the blood pressure reached its height the secretion of urine became nul under a simultaneous powerful contraction of the finer arteries of the kidney, and that with reduction of the pressure and returning relaxation of the vessel walls the secretion of urine began anew. Under the influence of digitalis the action of the heart is slowed down, the auricle gets time to empty itself because the systole sets in slower and later. So the diastole is longer and the ventricle gets its full supply. Thus, the stasis is relieved in all the distant vessels, the swollen mucosa of the bronchial tubes and the enlarged liver and spleen return to their natural volume, or are at least reduced, the cyanosis fades away, the dyspnœa disappears. As the pressure is increased in the arteries, including the arteries of the kidneys, the quantity of urine is increased and thus the dropsy is relieved.

These effects are seen in all cases of heart failure where the working capacity of the heart is diminished. Therefore digitalis is not indicated in cases in which the compensation is already perfect.

But digitalis has only a temporary effect, though it may be sustained for three or four weeks. Under unfavorable circumstances the effect is lost in the course of one or two weeks. The remedy should not be resumed too soon. It is best to allow an interval of four weeks, but the effect should be as perfect as possible that it may be sustained for some time. Digitalis has its full effect only when the patient remains in bed, and compensation is secured and sustained only when the patient remains in bed four weeks. Every lengthening of this period is a gain for the patient. In these cases where compensation is long sustained and again lapses, it may be restored at times without resort to digitalis simply by remaining in bed a few days (Liebermeister).

Digitalis is given in the form of the tincture, the infusion, and the powder. Some of the active principles are more soluble in water, some in alcohol; the powder of course includes them all. The dose of the powder is one grain; the equivalent of the tincture is ten drops, and of the infusion half an ounce, that is, one tablespoonful. The preparation may be given every two to four, or six to eight, hours. Any preparation used should be fresh.

Digitalis is peculiar in the fact that its action is slow, but is long sustained and is cumulative. Thus Pech finds that the administration of digitalis in small dose, 0.10 gm. (gr. iss.), makes its effect manifest only after four to five days. After the use of 0.20 (gr. iij.) the effect is seen in three days. After getting a permanent effect the remedy may be used a long time, but never longer than a

month. After the use of 0.30 (gr. ivss.) the effect is produced in twelve hours and continues for about five days. If this dose is continued five to six days, the after-effects are sustained for twelve days. A favorable effect is sustained for twenty days after the use of 1 gram (gr. xv.), but such large dose should never be administered longer than two days. The quantity of digitalis that may be taken in time without injury is sometimes enormous. A unique case in this regard was reported by Bälz. A woman affected with mitral stenosis of high degree took daily 0.3 gram (grs. ivss.) of the powder of the leaves of digitalis, and consumed thus in the course of seven years over 800 grams (℥ xxv.) of the drug. In the fatal case reported by Köhnhorn, that of a young soldier who took 100 pills (4 per day) to escape military service, it was calculated that the patient had taken in the course of four weeks 16 to 17 grams of the powder of digitalis, corresponding to about 0.6 (gr. ix.) per day. As 1 gram (15 grs.) is the highest maximal dose, it was seen there was in this case a fatal cumulative effect. Large doses of the tincture, as much as half an ounce, are sometimes prescribed in delirium tremens, and continued drachm doses in the precritical periods of pneumonia (Balfour). But such doses are dangerous in heart disease, as the vagus is paralyzed in time by the cumulative effects of even small doses.

To avoid the evils of cumulative effects, Pfaff recommends a preparation from which the digitalin has been extracted. This drug has a bitter but not disagreeable taste and is quite willingly taken by the most fastidious patient. It is absorbed much more slowly from the stomach. It was used with benefit in sixteen cases at the clinic at Basle. The largest dosage used was 48 mg. in twenty-four hours. Robinson finds that he is able to neutralize the effect of digitalis in contracting the vessels by the simultaneous administration of nitroglycerin.

In whatever form, the remedy should be suspended when the urine increases to two or three pints, or when the frequency of the pulse is reduced, or in the presence of unpleasant symptoms, vomiting, etc. During the interval the remedy should not be given at all.

Digitalis was used in the years 1870–1890 inclusive in the clinic at Tübingen in 94 cases of valve lesions with broken compensation. It was used also in 30 other cases in which the expectant method furnished no results or at least no satisfactory results. Of these 124 cases the result was perfect and permanent in 61, temporary in 40, and negative in 23 cases (Liebermeister).

The symptoms of poisoning by digitalis consist in almost unin-

terrupted vomiting, with violent headache, tumultuous action of the heart, and icy coldness of the extremities.

In the treatment of poisoning, it is necessary to wash out the stomach perfectly clean. The collapse which follows the poisoning is then to be treated in a purely symptomatic way by the proper stimulants, alcohol, camphor, caffeine, salt-water transfusions, etc. It is necessary to keep the skin warm by clothing and other methods (Schultz).

The efforts of the chemists to separate an active principle have succeeded in isolating a number of alkaloids, to wit, digitalin, digitonin, digitalein, and digitoxin. Schmiedeberg found the various preparations of digitalin to be different bodies. In fact most of these preparations are unreliable and unsafe.

The individual constituents of digitalis cannot be used with safety. The danger of intoxication, the impossibility of reckoning the proper dose of digitoxin—which almost caused Koppe, the discoverer of it, his life—moreover, the varying quantity of the constituents in different preparations and the varying susceptibility of different patients, make the use of these preparations impracticable (Oefele). In cases where digitalis disturbs the stomach, it is interesting to know that some of the constituents may be used by clyster. Thus Wenzel finds digitoxin (Merck) the most powerful as well as the most poisonous of all the constituents of digitalis. This author used it in nine cases, three of valvular disease, three of myocarditis, and three of nephritis. The digitoxin was used by clyster in the preparation: digitoxin 0.01, alcohol 10.0, aq. destillat. ad 200.0. Fifteen grams of this preparation were injected with 100 gm. water three times a day, thus of digitoxin 0.00075 per dose. The effect in strengthening and retarding the pulse, increasing the diuresis and relieving the stasis, without any associate evil, was more powerful than with the use of the infusion of digitalis (Dippe).

It seems to be established that digitalin may cause a local inflammation and even necrosis of tissue when used subcutaneously. Watkowski and Pell saw a sharp congestion at the point of penetration, which was attended with fever. Hence Erlenmeyer warns against the use of digitalin by subcutaneous injection.

Better adapted for this purpose is sparteine, which is highly soluble and not at all irritant to the tissues. The sulphate of sparteine is given in the same dose as morphine, namely, one-sixth to one-fourth of a grain. Sparteine has an advantage also in that it does not increase the resistance in the vessels. Hürthle found from his studies that oxysparteine, administered in doses which

produced no visible change in the general condition, increased the activity of the heart without producing any change in the tone of the vessels.

Probably the best single substitute for digitalis is strophanthus, which is given in the form of the tincture in the dose of five to fifteen drops, beginning preferably with the smaller dose. Strophanthus has the advantage that it acts at once, never irritates the stomach or affects digestion, and has no cumulative effects. But strophanthus does not really dispute the sovereignty of digitalis as a heart tonic. The effect of it is neither so substantial nor so sustained. The throne of digitalis is established by decades of ripe experience at the hands of the most cautious and critical observers. The claims of strophanthus are still on trial. Gottlieb declares that he has never seen so many patients who have lived for years with heart disease die suddenly under any other treatment as after the use of strophanthus. The physician and the relatives console themselves, he says, with the diagnosis of an apoplexy, but the accumulation of cases of sudden death may point to a direct effect of strophanthus medication.

It is wise to know a number of substitutes for digitalis, as the remedy must be changed from time to time, not only because of cumulative effects and tolerance, but because of irritant effects upon the stomach and digestion. Digitalis will sometimes produce a most intense anorexia, and if then further forced, nausea and obstinate vomiting. Patients often reach a point when they will fight the further use of it, and if the physician does not know how to change the drug he will find that the patient knows how to change the physician. The following are therefore some of the principal substitutes as given by Oefele:

Adonis vernalis, which has something of the effect of digitalis and tannin, given in concentrated infusion or in alcoholic extract gr. xx. ter die, or in the form of adonidin gr. i. dissolved in glycerin and water ʒ iss., of which the dose is ten drops three times a day.

Cereus grandifloris, sometimes unbotanically known as cactus grandifloris, furnishes at the moment of dehiscence of flower a cardio-tonic preparation. The preparation is found in the plant only for a few hours, but from it is derived a fluid extract and a tincture, which are prescribed in the dose of eight to ten drops four times a day. The dose should never exceed two grams every four hours. This drug has no cumulative effect. Fletcher Home recommends cactus grandifloris in the treatment especially of nervous affection of the heart. This remedy was also recommended by Engstav and Bomet.

Convallaria majalis, lily of the valley, has been long employed by the people in the treatment of dropsy, especially in Russia. The

active principle is derived from the unopened buds. The infusion is given in the concentration of 1–10, of which the dose is a tablespoonful every two hours. Convallaria combines the principles of digitalis and jalap. Convallarin is not suitable for long medication, as tolerance occurs as early as the fifth day and the effect ceases from that time on, unless the dose is increased. In this regard convallaria is allied closer to the double caffeine salts than to the other digitalis bodies.

Hellebore is one of the drugs of the most remote antiquity. Helleborein possesses drastic and expectorant properties. It has, therefore, something of the virtues of ammonia and digitalis, and was employed in older patients affected also with bronchitis and constipation. Helleborein is easily soluble in water and may therefore be used in subcutaneous injection in dose of one centigram.

Nerium oleander, originally an arrow poison used by the aborigines of Spain. As an arrow poison it ranks in its rapid effects with strophanthus. The oleander possesses cardio-tonic glucosides very close to those of digitalis, so close that they have sometimes been regarded as identical. But they are not identical. The common preparation is an infusion. Merck made a tincture, Bombelon a preparation, an oleandrid, for subcutaneous injection. The effect of the remedy is prompt and sustained, as the pulse is kept regular and powerful for a long time. The frequency of respiration is reduced. Under the action of it there is diuresis with increase in the solid constituents of the urine. Constipation is relieved, and palpitation, œdema, and dyspnœa, the consequence of valve lesions, disappear. The daily dose for short, quick effect is 0.5 of the crude drug, or 5 grams of the tincture; for longer use 0.05–0.1–0.2 gram of the crude drug. For subcutaneous use the first dose should be oleandrid 15 mgm. dissolved in water and glycerin. This dose may be followed by 6 mgm. after an interval of forty-eight to ninety-six hours. The oleander toxin, which has also been recommended, is less suitable.

Euonymus atropurpureus and Rhamnacea, from North America, has as yet found no foothold in the old country, perhaps on account of the uncertainty of the drug. The cardio-tonic glucoside, euonymin (Meyer) produces no local irritation in subcutaneous use and sustains the effect of digitalis for nineteen days, but the internal administration of the crude drug irritates the intestinal canal to such an extent as actually to conceal the effect upon the heart.

Zea Mais is said to possess in its stamens, according to Italian observers, cardio-tonic properties and to act as a powerful diuretic. It is considered of especial value in catarrhal inflammatory affections of the urinary apparatus.

It is interesting to know that there is here a tropical and subtropical family of 103 species and 900 varieties, some of which may be found to contain the proper constituents to stimulate the heart with safety.

In seeking a substitute for digitalis it is well to know also that the remedy which acts upon the heart may have other properties. Thus adonis is constipating, convallaria is purgative, oleander is slightly laxative, hellebore is expectorant, and mais has anti-catarrhal virtues. Convallaria, like the double salts of caffeine, exercises a momentary effect and develops a rapid tolerance, so that it must be given in increasing doses like arsenic. Digitalis, oleander, strophanthus, adonis, in small dose often show their peculiar effects upon the heart only after a longer time. In the case of strophanthus, old age with its fragility of the vessels is a contra-indication on account of the danger of apoplexy. But digitalis and oleander may be taken with safety by the oldest patients. The attempt to find a substitute for digitalis in any one remedy, which has all its virtues and none of its vices, will perhaps always prove futile. Nevertheless, a successful therapy may be obtained by a rational change of the substituting drugs (Oefele).

In all forms of valve lesion associated with arteriosclerosis, that is, especially in aortic lesion, the sodium iodide is indicated in the dose of gr. v.–x. ter die in a wineglass of milk. The remedy may be given continuously with benefit in the smaller dose for months or years.

It will be remembered also that for steady support of the heart no remedy equals strychnine; but the virtues of strychnine have been sufficiently emphasized elsewhere.

The Fat Heart.

The accumulation of fat about the heart is a sign so obvious as to be actually obtrusive upon exposure of the heart; hence the fat heart was noticed by the oldest observers. In fact the increase in the normal fat was remarked as long ago as by Bonetus, Morgagni, and Sénac. These early observers noticed the deposit at the base of the heart under the pericardium and along the furrows formed by the vessels, especially in advanced maturity and old age. Lancisi (1738) saw also the spots of fatty degeneration on the surface of the heart muscle under the endocardium, ashy gray points which he distinguished as "sub forma cinerearum macularum sparsim internas tunicas variegant." Gambarini considered these yellow spots, which he found most frequent on the papillary muscles of the left ventricle, as

signs of myocarditis. But it was Laennec who first of all distinguished the mere deposit of fat from true fatty degeneration which represents a particular form of retrograde change in the heart muscle. We may now distinguish these two forms as the fat heart and the fatty heart, respectively.

The outside accumulation of fat, the simple fatty deposit (fat heart), was especially studied by Stokes, who described the condition as an envelope of yellowish color, which sometimes enclosed the whole heart, concealing the muscular structure in the vessels and enlarging and changing the shape of the heart more to that of a globe. It is plain to see that the accumulation is a simple increase in the natural fat, for the first increase is found in the regions of natural deposit, to wit, along the furrows between the ventricles and in the course of the coronary vessels, as well as between the auricles and ventricles, at the apex and at the base about the origin of the great vessels. The fat increases first to cover and conceal the right ventricle, while parts of the left ventricle are still uncovered. In its further course the fatty deposit extends to infiltrate the muscular structure, so that lines or stripes of fatty tissue are intercalated between the muscle fibres. Under the continued accumulation, the heart is at last loaded down upon its surface, and the muscle fibres are themselves separated, and finally suffer atrophy from pressure and defective blood supply.

These excessive accumulations are found usually in connection with general obesity and are distinguished in their local deposits as obesitas cordis or lipomatosis cordis. From the fact that the fat forms an envelope for the heart, Virchow described the condition as lipoma capsulare cordis. But, as in general lipomatosis, the deposit of fat may be localized, so also considerable accumulations are encountered in the heart in the absence of excessive deposits elsewhere. In fact, the fat heart is sometimes found in lean people, occasionally in individuals undergoing emaciation from some wasting disease. Accumulations of fat about the heart are occasionally found also with accumulations of fat in other internal organs. Laennec observed in these cases the accumulation of a large quantity of fat in the inferior mediastinum, and especially between the pericardium and the pleura. This fat, he says, is often traversed by a large number of small vessels which gives it a reddish color, and as the fat protrudes in the form of an irregular fringe into the pleural sac, it has a coarse resemblance to the cock's comb. "The fat which envelops the heart, on the other hand, is always of a pale yellow color and of a mediocre consistence." Moreover, in some cases fat comes to be deposited in the heart itself in unusual places, as, for

instance, under the endocardium, where it may form a layer, or more frequently accumulate in masses which may protrude into the cavity of the ventricles. Lancisi distinguished these deposits as adipose follicles.

Where the accumulation is excessive, the heart muscle itself suffers degeneration of various kinds, chiefly fatty, so that in these cases the two forms, the fatty deposit and the fatty degeneration, coincide. The accumulation is sometimes rapid, but is usually slow. Moderate accumulation in no way disturbs the action of the heart. The increase becomes pathological only when it interferes with the circulation.

The *etiology* in these cases is the same as that of obesity in general. But the fat heart is never found in infancy; it occurs only after maturity, and the tendency to it increases with years.

Obesity may be hereditary or acquired. The condition certainly runs in families. It is also more marked in certain races and places, and may be acquired by excessive nutrition and deficient exercise. Of foods, starch, sugar, and alcohol especially contribute to the formation of fat, so that the fat heart is often found in drinkers. A sedentary life, indolent habits, prolonged sleep, bring about the same result by diminished consumption.

No particular influence is exercised by sex. Most of the monstrous cases of general obesity have been in women. "Fair, fat, and forty," is a proverb. Bizot found the fat heart more frequently in the female sex, but Quain saw it oftener in the male sex.

Aside from the cases in which the local accumulation is part of a general polysarcia, which may in turn be ascribed to heredity, alcoholism, etc., it is difficult to account for the fat heart. The tendency of the present time is to refer these grave disturbances of nutrition, along with acromegaly, giantism, etc., to obscure changes in the central nervous system. The affection of the thyroid gland, which leads to myxœdema, probably has a similar central origin. The chief danger of lipomatosis arises from atheroma of the arteries and hemorrhage in the brain. The frequency of arteriosclerosis has long been remarked in connection with the fatty heart. In more than one-third of the cases of obesity the heart is found fatty, and a third of all the cases succumb to cerebral hemorrhage.

Symptoms.—The fat heart may show no signs. The amount of fat in general which may be consistent with health varies with different individuals and in the same individual in different periods of life. People gain and lose weight, which is largely a matter of the amount of fat, within certain but indefinite limits, without interference with comfort or health. It is questionable if any amount of fat upon the

surface of the heart may really embarrass its action. It is only when the heart muscle fibres are separated by layers of fat that the action of the heart may be impeded. The muscle of the heart is so strong when sound as to be able to overcome much greater obstacle than can be offered by any deposit of fat. For instance, the action of the heart is powerful and the pulse may be strong at the wrist in the presence of a large pericardial effusion. It is, therefore, not the accumulation but the infiltration, especially when followed by subsequent degeneration, which interferes with the action of the heart. Laennec declared that neither he nor Corvisart had ever observed any symptom "which might seem to depend directly upon this accumulation of fat." It is when the deposit is associated with degeneration that the action of the heart becomes enfeebled, so that the impulse may be barely perceptible to the touch or ear. The pulse is correspondingly weak and is sometimes, though rarely, retarded (bradycardia).

In connection with general obesity the fat heart is found in two conditions, the plethoric and the anæmic. The plethoric form is more common in men. It is distinguished by fulness of vessels with stagnant circulation, redness of the face, distended pulse, hyperæmia of the liver and lungs, hæmorrhoids, increase of the hæmoglobin and of the number of red blood corpuscles. The anæmic form occurs especially in women and shows itself in opposite conditions—in pallor of the surface and of the visible mucosæ, in weakness, languor, neurasthenia, palpitation, dyspnœa, œdema. The pulse is quick and feeble. The blood shows diminution of hæmoglobin and reduction in the number of blood corpuscles. Transition forms occur in both sexes.

The signs on the part of the heart are those of heart failure, which, with the prognosis and treatment, will be described in connection with the true fatty degeneration of the heart.

Fatty Degeneration of the Heart.

The fatty degeneration of the muscular fibres of the heart was first differentiated from mere accumulations or deposits of fat, under the critical observations of Laennec. This fatty degeneration Laennec described as a conversion of the muscular tissue into a substance which presents the physical and chemical properties of fat. It is a degeneration, he said, like that which was observed by Haller and Vicq d'Azyr in the muscles. When muscular tissue so affected is pressed between two layers of paper it makes them distinctly greasy, and it is in this way that the fatty degeneration may

be distinguished from a simple softening. Rokitansky, with Williams, Paget, Quain, and Stokes, devoted particular attention to the study of the fatty heart, but the finer histological changes were studied especially by Virchow, Zenker, and Wagner. Laennec had already mentioned the pale-yellow color, which he compared to that of the "dead leaf," but had observed that the degeneration was limited only to the surface of the heart, or proceeded from without inwards so that the muscular texture of the interior could be distinctly recognized, while it became gradually degraded toward the surface to the consistence and color of fat at the apex of the heart. This is the appearance which is presented to macroscopic inspection, and the apparent limitation of the process gave rise to the belief that fatty degeneration was rare and comparatively insignificant.

Microscopic studies, however, have shown that the heart may present a perfectly normal appearance to the naked eye, while the muscle fibres may show extensive degeneration. In fact, no degeneration of the heart is so treacherous in its general appearance. The process may be diffuse and widespread. It is generally, however, more or less localized or disseminated throughout the structure of the heart, and is most frequently found in association with other degenerative processes, as with the hyaline, fibroid, and atheromatous change. In fact, the association is so common that it has become a question whether we are justified in considering fatty degeneration as a separate affection. Fatty degeneration certainly goes along with and sometimes constitutes a prominent element of the various changes of myocarditis, and more or less fatty degeneration is found in the subsequent course of hypertrophy, assuming especial prominence in the stage of dilatation. The fatty degeneration which results from hypertrophy from any cause is explained in this way. So long as the hypertrophied muscle is able by increased work to meet the increased demands, more fat is consumed and there is no accumulation of fat in the region where the greater work is done. But if the muscle is innervated to greater work and the proper direction in metabolism is given to the increased destruction of fat, while the muscle itself becomes incompetent to overcome the obstacles in its way, part of the fat remains unconsumed and the muscular tissue is gradually substituted by fatty matter. In this way is explained the fact that the fatty degeneration occurs earliest and is most marked in the region of greatest work, that is, in the left ventricle (Leube).

Moreover, some degree of fatty degeneration is found in all cases of affection of the endocardium and pericardium. It is questionable, therefore, if fatty heart, strictly speaking, is still entitled to a separate consideration. The tendency of the present day is to consider

the fatty degeneration as a part process of myocarditis, and to give it no separate consideration as a distinct disease.

Nevertheless, it is true that in many cases the fatty is the principal form of degeneration. But the fatty degeneration is to be regarded in no sense as an individual disease, and the discussion of the subject in a separate chapter is a deference to a view which has become so widely disseminated as to be eradicated with difficulty.

Though this process may not be dissociated from the other retrograde changes of myocarditis, fatty degeneration is quite as frequent as is commony believed. Most of the cases of so-called parenchymatous degeneration are really cases of fatty degeneration. Thus Roemer had under treatment in the clinic at Tübingen in twenty years, 1870–1890 inclusive, 291 cases of valve lesion, of which 230 showed disturbance of compensation. At the same time there were 234 cases of myopathic heart failure without any lesion of the valves, every one of which showed the same symptoms of broken compensation as in the case of the valve lesions. As most of these cases were really fatty degenerations, it is seen that the condition is nearly, if not quite, as frequent as valve lesions, and the fact that the rupture of compensation in the case of valve lesions themselves depends chiefly upon this degeneration is further proof of the great frequency of the condition.

Krehl contends that the degree of fatty degeneration in certain parts of an organ cannot be determined, either macroscopically or microscopically. It is only by means of chemical quantitative analysis that the amount of fat in a muscle may be appreciated. Parts of the left ventricle and the papillary muscle which contain no interstitial fat are, after desiccation, treated with ether and the amount of lecithin is then estimated. The amount of lecithin varies in different cases independent of the variations of fat and water. The amount of fat in the heart varies within wide limits, even in people in whom the activity of the heart has shown itself to be normal up to a few hours before death. It is somewhat increased, as a rule, in pernicious anæmia, malignant neoplasm, and permanent tuberculosis, but is considerably increased, as much often as two to three times, only in phosphorus-poisoning. On the other hand, disease of the myo- and endocardium attended with weakness shows the amount of fat in the rule either normal or less than normal. At any rate, there is no correspondence between the amount of fat and the disturbance in the action of the heart.

Goebel, of Zurich, protests against this view of Krehl that only the chemical analysis may give an accurate estimate as to the degree of fatty myo-degeneration. Goebel remarks upon the fact that

Krehl omitted the layers of heart tissue near the endo- and epicardium, in which the fat formation is often most marked. Goebel took pieces from the most different parts of the heart and examined them *always fresh*, without and with acetic acid and potash solution, and thus examined fifty-eight hearts in different affections, heart disease, lung disease, sepsis, carcinoma, atrophic kidney, tuberculosis, diphtheria, etc., finding always evidence of more or less extensive fatty degeneration.

Flexner also contends against the point taken by Krehl as to the necessity of chemical analysis, maintaining that a careful microscopical examination will determine the condition. Flexner finds in a series of diseases, especially in the infections, fatty degeneration of the heart muscle up to the most extreme degree in which scarcely a cell is spared. Still more conclusively, Flexner was able to produce fatty degeneration of the heart muscle in guinea-pigs and rabbits by the injection of diphtheria cultures, dog serum, abrin, and ricin, and believes that the fatty degeneration of the muscle-cell protoplasm depends upon the action of toxins.

Morbid Anatomy.—Where the degenerative process is diffuse and extreme, the conversion of a part of the heart substance into fatty matter may be recognized with the naked eye. The muscular tissue loses its fresh red color, and becomes brownish or yellowish red. This transformation is, however, very rarely general; it is usually localized and is most marked about the apex. It is seen after removal of the superjacent fat, which is often increased in amount, that the muscular tissue is fatty in places, but that even in these regions, as Laennec observed, the fatty matter does not penetrate the depths of the structure, but is largely disposed about the surface. The degenerative process is most marked in the left ventricle, and particularly in the region of the apex. The degeneration next affects the septum, and later, in less degree, the right ventricle. The auricles suffer least of all. But the examination of sections at various depths discloses the fact that the limitation of the degenerative process to the surface is more apparent than real, as it is seen under the microscope that the parts which really suffer most are the papillary muscles and the tissue that constitutes the trabeculæ. It is in these regions that, when the process has become extensive, the evidence of fatty degeneration is visible to the naked eye as a collection of yellow masses under the endocardium. Brault saw insular masses of fatty degeneration in healthy muscle in two cases of ulcerative endocarditis. The small depots which were most pronounced in the papillary muscles stood out with their yellow-gray color in sharp contrast to the brown red of the surrounding healthy tissue. The heart muscle thus

presented a peculiar mottled appearance. Accurate analysis with osmic acid showed that the masses lay in the midst of healthy tissue. The vessels were perfectly normal. There was no infarction.

Goebel found that (1) fatty degeneration of the heart occurs usually in masses and becomes diffuse by dissemination and gradual coalescence, but in such a way that spots of most intense degeneration may be still marked; (2) The fat drops are found in the interfibrillary sarco-neoplasm and are thus arranged in rows; (3) Striation does not essentially suffer in fatty degeneration, but is thrown in the background in the presence of the fibrillary accumulation of fat drops, especially under treatment by acetic acid; (4) Segmentation often occurs in connection with fatty degeneration, especially in the beginning of the process; (5) The fatty degeneration seldom stands in direct relation to the dilatation and hypertrophy of individual sections of the heart, for when one chamber is hypertrophied, both sides may show fatty degeneration; (6) General fatty degeneration in consequence of toxic action is rarely uniform, but is more marked in certain places; the layers under the epi- and endocardium show the most pronounced fatty degeneration.

Conclusive evidence of fatty degeneration is furnished only by examination under the microscope. The sections show, first, that the fatty matter is intercalated between the muscular fibres. The fat shows itself in rows or drops disposed like a string of beads between the fibres of the heart muscle. Where the deposit is extensive the fat globules apparently constitute the bulk of the tissue. But the process is not limited to a mere infiltration. The fibres themselves suffer degeneration. It is seen that the striation has become faint or disappears. The muscle fibre appears granulated and opaque, but the opacity clears up under acetic acid. A little later dark highly refracting granules appear, arranged in lines or rows, and these granules gradually take the place of the muscle fibre. Under a little higher power it is seen that these granules are oil globules, and associated with them are separate bodies of dark color, which are recognized to be pigment granules, which impart to the fibre a darker color. In the course of time the muscular tissue itself is largely replaced by these minute oil globules, which change the consistence of the tissue, rendering it lax, flabby, and fragile. The muscular structure has now changed color. It is more of a yellowish or ashy color, or under the accumulation of much pigment matter assumes a mottled yellowish-brown or brownish hue.

Besides these changes there occurs a peculiar separation of the muscle fibres, which, instead of being firmly connected, show now distinct interspaces. It was Renaut who called attention to this disinte-

gration or dissociation of muscle fibres. At times a somewhat analogous process is seen, in which the muscle fibres are separated not only along the line of union but also in their length; this process has been elsewhere described as a fragmentation. How far these processes of dissociation and fragmentation may contribute to the heart failure is not yet determined, but the chief factor must always remain the substitution of the intensely active protoplasm of the muscle fibre by the inert fatty matter.

Fatty degeneration of the muscular tissue is commonly found in association with other affection of the myocardium. The occurrence of fatty degeneration in myocarditis is usually distinguished as a regressive metamorphosis. Sometimes there is hypertrophy, sometimes atrophy. There is always more or less atheroma of the coronary vessels, sometimes amounting to thrombotic occlusion with centres of softening, so as to constitute myomalacia. In other cases there is distinct evidence of the coincidence or the precedence of pericarditis or endocarditis. Fatty degeneration of the heart is also found in connection with fatty degeneration of the vessels of the brain, not only in the aged, but also in the young, and often without the evidence of fatty or atheromatous change in other arteries of the body (Friedreich). In a case recorded by Rolleston the fatty degeneration clearly began in the epicardium and was confined to the right ventricle which was united to the pericardium, while the left ventricle, hypertrophied from a light mitral insufficiency, showed perfectly normal musculature.

Whether free fat may ever be absorbed into the blood from the heart is a question still unsettled. Smith, as long ago as 1836, found globules of liquid oil in the blood, and Dumesnil and Pouchet, afterwards Magnus Huss, made the same observation. But Parrot thinks that the oil was liberated at the autopsy under section of the myocardium, and that a certain quantity of fat infiltrated in the muscle became mixed with the blood in the cavities of the heart (Petit). Cohn found in one case fine crystalline stellate needles of fat in the midst of the muscular tissue.

When the fatty degeneration is at all diffuse it leads necessarily to dilatation, so that the fatty heart is distinctly enlarged. Boettcher and Valentiner both observed an absolute increase of fat in the course of fatty degeneration. Boettcher found the increase from two to four per cent., while Valentiner, who compared the tissue of the normal heart with that of the hearts of drinkers, which did not appear to be affected by fatty degeneration, found an increase of fat in the drinker's heart of from one-half to one per cent. A warm knife looks greasy or oily after having been used to cut a fatty heart.

Etiology.—The fact that the fatty degeneration rarely occurs in infancy or early youth points to the influence of disturbance of nutrition, especially of the walls of the vessel, as the initial lesion. Hayden found in eighty-eight cases that fatty degeneration was most prevalent between the ages of sixty and seventy, and next between forty and fifty. Confirmation of this view is found in the fact that the fatty degeneration affects chiefly the muscle fibres, which have the most to do or are subjected to greater pressure or tension. Quain called attention to the frequency of atheroma of the coronary arteries in fatty degeneration of the heart, and showed that the regions affected correspond to the domain of the contracted or obliterated vessels. He found calcification or obturation of the coronary arteries thirteen times in thirty-three cases of fatty degeneration, and compared the condition with the softening of the brain which takes place under similar circumstances. Markham saw degeneration of the coronary arteries in every one of twelve cases of fatty degeneration of the heart. Leyden and Stoffela corroborate these findings in recent studies. In the face of these observations, which might be largely multiplied, it is difficult to understand how Wilde refuses to admit any direct relation between lesions of the coronary vessels and fatty degeneration of the heart muscle. Everywhere is seen under defective nutrition a disposition on the part of protoplasm to fall back on the easier task of forming fat rather than to carry on the more arduous duty of manufacturing new material like itself (Foster).

The first defect in nutrition is a defective oxygenation. The theory is suggested that the interference with the circulation deprives the tissues of oxygen, so that the nitrogenous constituent of the muscle protoplasm is separated (together with the sulphur and phosphatic salts) and is absorbed from the blood, converted into urea and excreted with the urine (Fraenkel, Fleischer, Penzoldt). What is left is fat. Stoffela, too, considers a defective oxygenation of the muscle cell as the immediate cause of fatty degeneration.

Exquisite cases of fatty degeneration of the heart are found in connection with certain intoxications, especially by phosphorus and arsenic. These cases give a clew to the toxæmia of some of the acute infections; and the alteration of the heart muscle, which occurs in connection with various infectious diseases, especially with typhus and typhoid fevers, diphtheria, scarlet fever, septicæmia, puerperal fever, etc., gives evidence that the degeneration is due to the direct action upon the muscle fibre of some toxin. Brault believes that the condition is always the expression of a toxic infection, as in the case of fatty degeneration of the parenchymatous organs.

Fatty degeneration of the heart occurs also in the various forms of anæmia, especially in leukæmia, chlorosis, and progressive pernicious anæmia. Gusserow found fatty degeneration as the only recognizable alteration in the bodies of five patients who suffered from extreme anæmia in pregnancy, and who perished without exception before term (Schroetter). Fatty degeneration occurs also in the anæmia which is produced artificially by venesection. Perl found the fatty change in six of seven dogs, which had been repeatedly bled at short intervals. The degeneration was most pronounced in the papillary muscles of the left side, next in those of the right side, and then in succession in the walls of the left ventricle, left auricle, right ventricle, and least of all in the right auricle. Thus is explained the fatty degeneration which occurs after frequently repeated or long-continued hemorrhage, epistaxis, hæmoptysis, hæmatemesis, metrorrhagia, enterorrhagia from ulceration or hæmorrhoids, hæmaturia, etc. The particular degeneration may be attributed in all these cases to the development of a toxin. Poisons are also liberated in the process of inanition, and fatty degeneration is commonly found, in connection with other changes, in the marasmus and cachexia of phthisis and carcinoma.

Fatty degeneration is found, further, as stated, in the various affections of the pericardium and endocardium. Thus Wagner found in 2,000 autopsies, 35 cases of severe pericarditis, and in 17 of these cases microscopic examination revealed fatty degeneration of the heart substance. Affection of valves was found in 75 cases, and of these, 28 showed the evidence of fatty degeneration. The peculiar toxæmia which produces acute yellow atrophy of the liver leads also to fatty degeneration of the heart, the process in both cases being a kind of rapid fatty necrosis, which, as in the case of the acute poisoning of phosphorus, extends also to involve other organs, especially the kidneys. Fatty degeneration of the heart sets in also in the later course of Bright's disease and gout, contributing largely to the gravity of these affections. Charcot attributed the fatty degeneration which occurs in gout to the changes in the coronary vessels in this disease.

The degeneration of the heart which occurs under the abuse of alcohol is also chiefly fatty. Abundant reference to the changes produced by alcohol has been made already in the discussion of idiopathic hypertrophy and myocarditis. Here it may be remarked only that it is the habitual rather than the excessive drinker who suffers from the fatty heart. Beer is the form of alcohol which is principally responsible for fatty heart, partly because of the quantity of fluid which overloads the vessels and increases the work of

the heart, but chiefly, it is thought, on account of the potash salts which directly poison the heart muscle. Thus fatty degeneration of the heart, which occurs usually after the meridian of life, is not infrequently encountered in Germany in students who indulge in large quantities of beer.

Fatty degeneration of the heart is certainly found most frequently in the male sex. Ormerod gives the proportion as eighteen to seven.

There remain to be mentioned the effect of high living, "luxus-consumption," and over-strain from hard work. These two factors have already been invoked to account for the development of idiopathic hypertrophy. The disturbance of compensation, which finally ensues in all cases, depends largely upon substitution of the muscular tissue with fat. Thus fatty degeneration occurs at both ends of the social scale; in the high-livers of the upper walks and in the hard workers of the lower walks of life. The successful merchant, the rich banker, or the professional man who has retired upon a competency, finds that his comfort is disturbed by palpitation and dyspnœa, while the hard-working man is sent to the hospital or is cut down before the end of his days, with the signs of heart failure. But it is not only the hard workers, porters, boatmen, founders, "the hewers of wood and drawers of water," who suffer from over-strain. A number of cases occur among soldiers under the exhausting drills of modern discipline and after long marches in active campaigns, and quite a respectable contingent of cases is furnished in the modern gymnastics and sports, bicycle riding, boat rowing, ball playing, etc. Stout persons and lean ones alike die of fatty degeneration of the heart; and it may be found in every rank and position in life, from the millionaire to the bricklayer's laborer (Fothergill).

As in the case of myocarditis and hypertrophy, of which the fatty metamorphosis is only the later stage, the degenerative process may set in gradually, or may begin suddenly under a single excessive over-strain. Quain called attention to the effect of emotions of depressing character, especially grief, disappointment, or failure in any field, upon the nervous system and indirectly upon the circulation. The broken hearts are always cases of myomalacia under defective nutrition. A sound heart never breaks.

Symptoms.—The fatty heart is often latent. It is really surprising to what extent the heart muscle may suffer fatty degeneration and yet show no sign of failure. In a case recorded by Leube, the whole apex of the heart was converted into a mass of fat without the exhibition of a single symptom during life. It will be remembered that the heart has enormous reserve force. Wagner has shown that the preservation of a few healthy fibres suffices to sustain the heart

in the performance of its functions. The time of the occurrence of symptoms will depend in general on the demands on the heart. In a quiet life the signs of heart failure may show themselves late, or not at all. On the other hand, where the life is spent under anxiety and strain or in the efforts of hard work, the signs of degeneration show themselves sooner. Sometimes the break-down is sudden. The heart does its work regularly for a long time and then gives way all at once. The sudden deaths under the stimulus of the emotions, the deaths under the influence of anæsthetics are explained in this way. It will be remembered that the degeneration of the heart muscle may be excessive, but may show no evidence to naked-eye inspection. It must be appreciated also that muscular tissue may fail in its function independent of a degenerative process. But the more searching investigations of modern times usually disclose lesions corresponding to the degree of heart failure. Attention may here be again called to the studies of Krehl, who found lesion of the heart muscle in every case of valve disease where the substance of the heart was cut into sections and subjected to thorough study.

While it is true, therefore, that fatty degeneration in light degree may escape recognition, it is also true that any advanced degeneration makes itself manifest by the distinct signs of heart failure. It is, however, not for a moment to be maintained that the heart failure of fatty degeneration differs in any way from that of other degeneration, the fibroid, waxy, amyloid, hyaline, etc. The result in destruction of muscular tissue is the same in all cases. Nevertheless, there are certain signs which point more distinctly to this process. The fatty degeneration sets in, as a rule, slowly. The patient begins to complain of the signs of heart failure at first after exercise. There is a feeling of oppression in breathing. It is observed that the breath is short, and the heart palpitates upon exercise, as in climbing a hill or ascending stairs. The feeling is attended with a sense of constriction and distress in the region of the heart. Precordial distress and sensations of discomfort and oppression are frequent, but anything like real pain is rare and is probably due to complications. Thus both Friedreich and Quain saw cases marked by pain as severe as the attacks of angina pectoris, but these attacks were more likely due to the causative or coincident calcification of the coronary arteries than to simple fatty degeneration of the heart.

The pulse is usually increased, sometimes to 120 per minute, rarely reduced to 60, and in extremely rare cases (three times in one thousand cases) to 50 or even to 30. Such a retardation is a dangerous sign.

The sphygmograph shows in twenty-four per cent. of cases

increased tension as a sign of arteriosclerosis. Arhythmia exists in eight per cent. of cases, varying in degree from a simple intermittence up to absolute irregularity, which, as evidence of serious degeneration of muscular tissue, is a sign of great danger (Kisch).

Vertigo sets in early. The brain is the most sensitive of all organs and expresses the defective blood supply in unmistakable signs. Thus the patient is overcome by a feeling of faintness or vacancy of mind. Sometimes the feeling is attended with sensations of slight nausea, which pass away upon the assertion of the will or upon effort. In a more marked case the sensation may be attended with a temporary blindness or with an actual attack of syncope. Stokes laid great stress upon the pseudo-apoplectic attacks, which occur suddenly, last a variable length of time, are sometimes followed by actual paralysis, and may even terminate fatally in coma. Stokes speaks of cases in which the patients suddenly become comatose, a condition "which may be preceded by loss of memory and the lethargic state." He knew one case of this kind "in which on the occurrence of the premonitory symptoms the patient, by hanging his head so that it rested on the floor, used to save himself from an attack." The attacks of momentary vacancy and vertigo which thus occur are sometimes misinterpreted as preludes of epilepsy, or as actual seizures of the petit mal.

Sometimes the breathing is peculiar. It was Stokes, again, who first laid stress upon the irregular respiration which may occur in these cases during sleep; it is marked by a series of long-drawn inspirations, which gradually reach a maximum in length and depth and then gradually diminish until the breathing ceases entirely; after an interval, in which the patient seems to be dead, and which may excite the greatest apprehension and solicitude, the breathing again returns, at first slowly and superficially, and then gradually increases in strength, to be again interrupted as before. These peculiar phenomena were afterwards graphically described by Cheyne, and have ever since been known as the Cheyne-Stokes phenomena. They are known not to be pathognomonic of fatty degeneration, but to be due to any defect in the blood supply of the centre of respiration in the medulla oblongata. Thus typical Cheyne-Stokes respiration has been observed in cases of tumor of the brain, basilar meningitis, in uræmic coma, and in a grave attack of pericarditis. Fothergill quotes cases from the work of von Dusch, who saw this phenomenon in cerebral hemorrhage and in venous hyperæmia of the brain from tricuspid regurgitation.

Certain cases are marked by anæmia and failing strength, with occasional attacks of palpitation, which sometimes amounts to a veri-

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table attack of asthma. The degenerative process may be arrested or become quiescent at this stage, and patients may remain in this condition for years, suffering discomfort only under emotional distress or physical effort, the condition being not so necessarily progressive as in the case of some other forms of degenerative change. Any violent strain, more especially the intercurrence of some infectious disease, rapidly aggravates the condition and leads to the danger of sudden heart failure and death.

Physical Signs.—Light degrees of fatty degeneration are entirely unattended with physical signs. It is only when the cavities of the heart have become dilated that the physical signs are distinct. Inspection reveals a diminished impulse and a weakened or absent apex stroke. The reduction in the force of the heart may be more evident on palpation. When the fatty degeneration coincides with the general increase of fat, the action of the heart may not be perceived at all. Where the outlines may be made out by percussion, it is seen that the heart is enlarged and the increase of dulness will be observed to the right or left according to the ventricle which is affected. Thus hypertrophy of the right ventricle will be noticed in fatty degeneration of the left ventricle with dulness extending to the middle or right border of the sternum, while laxity of the left ventricle in the later course of the disease might be recognized by an increase of dulness beyond the mammillary line.

The heart sounds are weak. They may be perfectly pure, but they are often attended with the systolic murmurs of relative (muscular) insufficiency. Unless the degenerative process is confined to the left ventricle and is followed by consecutive hypertrophy of the right ventricle, there will be no accentuation of the second pulmonary valve sound. The murmurs characteristic of valve lesions are absent entirely, except in cases where the fatty degeneration occurs in the course of the hypertrophy which is consecutive to the valve lesions themselves. In extreme cases the dilatation of the right ventricle leads to relative insufficiency of the tricuspid valve, which is recognized by a systolic murmur at the ensiform cartilage and by the venous pulse in the neck.

The general signs of stasis supervene in the later course of the degenerative process, the dyspnœa increases, the pulse becomes feebler and quicker—is retarded in only the most exceptional case—and is so faint as to fade away when the arm is lifted from the body. Fluid now begins to accumulate about the ankles at night, and later to remain there during the day. The dropsy occurs at first as a local œdema. Where the accumulation has been so slight as to have escaped notice the physician finds that the skin over the tibia pits

upon pressure. The attacks of palpitation become more frequent. With dilatation there is a veritable tachycardia, later arhythmia and delirium cordis. By this time the general dropsy has become more or less pronounced. There is dyspnœa upon the slightest effort and attacks of difficulty of breathing occur in paroxysms. The patient may no longer lie down but must observe the semi-recumbent or upright posture.

The retardation of the circulation through the kidneys is indicated by the reduction in the quantity of urine, which is high-colored as well as scanty, and shows albumin and hyaline casts. The mind wanders, the patient becomes flighty under transitory attacks of uræmia. The presence of toxins in the blood develops the tendency to thrombosis. The blood clots in the heart, and emboli may be detached from the right heart to the lungs, sometimes causing sudden death by suffocation, or from the left heart to the brain with signs of true apoplexy. Most of the cases of sudden death are really due to fatty degeneration of the heart muscle, and may be ascribed to anæmia of the brain, in rarer cases to embolus of the brain, to acute œdema of the lungs, but much more frequently to sudden paralysis of the heart. Sometimes the death is due to rupture. Quain found sudden death 54 times in 83 cases, from rupture of the heart 28 times, from syncope (paralysis) 26 times.

Some of the symptoms on the part of the brain result from the defective blood supply and are expressions of cerebral anæmia, but other symptoms on the part of the brain, attributed to fatty degeneration of the heart, are more clearly due to the irritative changes in consequence of diseases of the blood-vessels of the brain itself. Thus Fothergill speaks of the querulousness, the fussiness, the caprice, the rapid variations of temper manifested by persons with fatty hearts, "the vacillating procrastination they exhibit, their inability to decide on matters, their unreasonable and inexplicable conduct, their whims, their preferences and dislikes, are all well known and are of diagnostic value." These signs certainly belong rather to changes in the brain cells themselves. Richardson speaks in a pathetic way of the feeling of incompetency which gradually sets in in these cases. "The man or woman with a hesitating heart is thereby unfitted," he says, "for sudden tasks, demands, or resolves, which, when the heart is firm, are considered of comparatively little moment. . . . From these circumstances some persons who once were known as resolute and determined lose those qualities when they are subjected to intermittent action of the heart, becoming, as they themselves feel and know, less the master of themselves and less secure in their own work and skill and power." Fothergill says

these persons seem often to be perversely stupid, and yet "if attentively observed they are seen to be doing their very best and are acutely and painfully conscious of their inability to take in what is said."

We have so many more reliable physical signs in our day as to render superfluous appeal to subtle psychical changes, which are often more demonstrative of the fine descriptive powers of the writer than of the nature of a disease.

Diagnosis.—The diagnosis is sometimes easy, but is usually difficult, and is occasionally impossible. The difficulty and impossibility of making a diagnosis becomes patent with the observation that the condition is often unrecognized, even at the autopsy. The diagnosis may be established in these cases only by a searching microscopic examination. The general diagnosis rests upon the signs of heart failure, palpitation, dyspnœa, venous stasis, cyanosis, dropsy, etc., together with the evidence of insufficient blood supply to the organs, cerebral anæmia, oliguria, uræmia, etc. There are no signs by which a heart failure from fatty degeneration may be differentiated from that due to any other cause. When the individual is past middle life, is corpulent, shows general evidence of fatty change, as in the arcus senilis, or of calcareous degeneration in the temporal and radial arteries; or if these signs show themselves in a drinker; if they set in slowly in the course of anæmia in any form, or after hemorrhage or other exhausting discharge; or rapidly in the course of a grave acute infection, the diagnosis of fatty heart may be entertained. But no one of these symptoms, and no array of them necessarily indicates fatty degeneration of the heart. The arcus senilis, the calcareous arteries, may be the sole signs of senescence, and the various symptoms of heart failure may be due to other degenerations or to incompetency from purely nervous cause.

The arcus senilis, to which prominence has been given among the changes of fatty degeneration, was first of all described by Canton, 1850. The arcus senilis is the visible segment of discoloration in the upper zone of the cornea. Canton described two distinct forms, one due to a deposit of lime salts found in healthy old people, the other a fatty degeneration found in connection with a feeble gait and irregular action of the heart. But the arcus senilis by no means of necessity indicates a fatty heart. It is often found in the absence of any fatty degeneration of the heart, and is itself absent in cases of well-marked fatty degeneration.

Great caution must, therefore, be exercised in the diagnosis of fatty heart, as there are no sure symptoms to indicate the existence of it, and the condition is not infrequently simulated in the weakened

heart which arises from the abuse of alcohol, coffee, tea, or tobacco, in the course of chlorosis and anæmia, or in consequence of bodily or mental strain. Hart reported two cases of cerebral affection simulating apoplexy dependent upon fatty disease of the heart.

Though the heart is usually overloaded with fat in obesity, yet this condition is not necessarily attended with fatty degeneration of the heart. On the contrary, many cases of extreme fatty degeneration are found in the emaciated, bed-ridden subjects of slowly advancing tuberculosis or of marasmus from any cause.

The diagnosis is really established by consideration of all the evidence, by the etiological circumstances, old age, alcoholism, infection, Bright's disease, etc., by the indications of fatty degeneration elsewhere, by the signs of heart failure, usually in the absence of valve lesions, and by the resistance of these signs to treatment.

In no case may the deposit of epicardial fat, which leads to atrophy of the muscle fibres, be distinguished from the true fatty degeneration in which the muscle fibres are replaced by fat.

Prognosis.—The prognosis is in a general way unfavorable, but depends in large degree upon the extent and rapidity of the process. The acute fatty degeneration which occurs rapidly in consequence of poisoning by phosphorus, oxalic acid, and more slowly under the influence of arsenic, together with the fatty degeneration which occurs in Bright's disease, tuberculosis, or pernicious anæmia, are often rapidly fatal. On the other hand, the limited process, showing but few signs of heart failure which disappear under rest and appropriate treatment, is not incompatible with a long life. The fatty degeneration of old age is not so unfavorable as might at first sight appear. In fact, some degree of fatty degeneration has been looked upon as a conservative process, lightening the heart to the lessening demands. Jenner once spoke of fatty degeneration of the heart as a *preservative lesion.* The heart acting with the full force of hypertrophy would easily break calcareous or atheromatous arteries in the brain, or even in its own structure. A certain degree of fatty degeneration relieves the damaged blood-vessels of extra pressure and thus obviates the danger of rupture. But the general fact that the fatty change in the heart goes along with degenerative changes in the blood-vessels and various organs of the body, makes the outlook for a long life unfavorable. There is, of course, at any time the danger of apoplexy from thrombosis or rupture of the cerebral arteries. The nutrition of the heart itself is impaired under the progressive changes of arteriosclerosis.

But the fact that the fatty heart does not take life rapidly, as a rule, is shown in the statement of Watson, who found the greatest

number of deaths in any single year at the age of sixty-three. In this consideration, however, it will be remembered that while fatty degeneration of the heart belongs, as a rule, to advanced life and does not begin to develop until after the meridian of it, the more searching microscopic investigations of modern times disclose the fact that most cases of myocarditis have fatty degeneration as the prominent or predominant lesion of the heart, and that myocarditis is especially frequent and fatal in the course of the acute infections (diphtheria, scarlet fever, typhoid fever, and rheumatism) of childhood and early youth. In this way may be explained the exceptional cases which are sometimes seen in early life. Thus Ward saw fatty degeneration of the heart in a child, and Dickinson reported a fatty degeneration of the heart in conjunction with an overloaded stomach, which was fatal at the age of three years.

Finally, the immediate prognosis is determined by the influence of the heart tonics. Where digitalis fails to tone the heart, the outlook in all cases is bad.

Treatment.—The fatty heart may be prevented or postponed and the degeneration may be limited by attention to the habits of life, by avoidance of exposure to the infections, by sparing the body overstrain and fatigue, and by control of the emotions. As the first signs of fatty degeneration are commonly noticed after some unusual effort or strain, it is not surprising to learn that the final catastrophe often occurs suddenly in this way. The number of sudden deaths at railroad stations which have happened to individuals on running to catch a train, the frequency of sudden deaths in water-closets under the effort of straining at stool, indicate the direction of prophylaxis. Though the older patient is protected by limited powers, the danger of sexual intercourse in all cases of weak heart must be borne in mind. Younger people affected with fatty heart will abstain from the gymnastic exercises which are carried to such extremes at the present day (ball-playing, boat-rowing, bicycle-riding). On the other hand, care will be taken to avoid the indolence which favors fatty degeneration from inactivity and disuse. As already intimated, the fat which is deposited should be consumed by the proper amount of muscle work. All bad habits which tend to the accumulation of fat, over-indulgence at the table, ingestion of alcohol, especially beer, should be surrendered. The drinks should be limited, that the vessels be not overstrained. A mixed diet is the best; simple plain food, in sufficient variety and small quantity furnishes the best result. Where there is much anasarca the quantity of fluid should be decidely limited. Balfour puts the patient affected with dropsy on the driest possible diet, and prescribes and limits the amount as follows:

Breakfast.—One single slice of dry toast, weighing about an ounce and a half, with no butter, but with a single cup of tea infused not longer than four minutes, with cream and sugar, amounting in all to not more than four ounces—and nothing else.

Dinner.—Not more than the lean of two chops, or its equivalent in chicken or fish; no vegetables; as much dry toast as may be desired; half an ounce of brandy, whiskey, or gin, in three ounces of water—and nothing else.

Supper.—As much dry toast may be taken as is desired, along with half an ounce of brandy, whiskey, or gin, in three ounces of water; and nothing more.

The patient is not allowed to drink much between meals, but if thirsty is permitted to sip slowly three or four ounces of hot water about an hour before each meal.

The writer has had no experience with this system, or with anything like an approach to a thirst cure, finding that an exclusive milk diet with confinement to bed and the administration of calomel in diuretic dose, three grains three times a day, generally suffice to dissipate an anasarca in less than a week. Where there is great ascites the cure is hastened by puncture with a trocar, or in bad cases by puncture and drainage of the lower extremities. Resort to these extreme means is justified only in exceptional, and for the most part neglected, cases, and even in these cases the dropsy may be dissipated by appeal to the infusion of digitalis, especially when fortified by the administration of ten to fifteen grains of potassium acetate with each dose. Upon the subsidence of œdema or the disappearance of other signs of heart failure, an attempt may be made to tone the heart muscle by regular graded exercise, as already described in the treatment of the valve lesions.

Sometimes the best prevention of fatty degeneration consists in the cure of anæmia by a proper ferruginous treatment or in the control of hemorrhage, as by curetting the uterus, nephrectomy for the relief of hæmaturia, some surgical intervention in the cure of hæmorrhoids, etc. Coats reported a case of fatty degeneration of the heart produced by the anæmia following a single large hemorrhage.

The obese patient may be put upon one of the approved formulæ (Ebstein, Oertel). The quickest results in reducing the weight and toning the heart are obtained at Marienbad, Austria, with regulation of exercise, hot baths, massage, and free libation of the alkaline mineral waters.

The diet, drink, and régime at Marienbad are arranged as follows:

Morning: five to six o'clock, three or four glasses (250 gm.) Kreuz or Ferdinand spring at intervals of fifteen to twenty minutes; then

a walk of one or two hours through the woods; hereupon, breakfast, a cup of coffee or tea (according to custom) with one tablespoonful of milk (no sugar), 50 gm. zwieback without sweetening or fat, 20 to 50 gm. cold lean meat or ham (without any fat), no butter.

Morning: ten to eleven o'clock a Marienspring bath with the addition of 2 to 3 kgm. soda, temperature 26° R., duration fifteen minutes, followed by a cold rain douche and cold rubbing; one hour's walk and then one glass (250 gm.) Wald (forest) spring with the addition of lemon juice.

Steam bath with cold rubbing, when the heart is sound and there is no arteriosclerosis, twice or three times a week.

Noon: one to two o'clock, a small cup of weak soup (without fat or starch, sago, bread, etc.); roast meat without fat or sauce, 150 to 200 gm.; vegetables, spinach, cabbage, cauliflower, and white bread 25 gm. For drink one to two wineglasses, according to custom, or even a half a bottle of good wine; no beer, no champagne, no liqueur.

Afternoon: seven to eight o'clock, roast meat, cold roast, or lean ham, 100 to 120 gm., bread, 15 to 20 gm.; after eating, walk of one hour. Cold washing of the whole body before sleeping. Sleep not longer than seven hours.

Even when not obese, the subject of fatty heart should be careful of the fat-producing substances. He should abstain largely from sugar and limit the quantity of starchy food. Where the case is at all marked the very best results are secured by the milk diet, and in the presence of œdema about the ankles, which is one of the first signs of insufficiency, the patient should not only be put upon an exclusive milk diet, but should be confined to his room. With the first signs of heart failure the patient should go to bed. By far the best results are secured in this way. Of all the remedies in the materia medica, no one furnishes results so satisfactory as the recumbent posture in bed.

Exercise should be undertaken later, but with extreme precaution and only, at first at least, under the supervision of the physician. The amount and character of the exercise will be determined by the immediate effects, but every exercise will on all occasions stop short of fatigue. There is greater danger of forcing the heart beyond its capacity in fatty than in any other form of parenchymatous degeneration, if only because of the coincident fatty and calcareous degeneration elsewhere in the body. The exercise at first should be, therefore, of the simplest character, and may consist simply of movements of the arms, after the manner of the exercises of children at school. In the course of a few days the patient may practice with some of the various forms of room gymnastics. The apparatus, which consists of iron plates of different weight to be lifted by ropes and pulleys,

may be easily adjusted in any room. The exercise may then be varied with Indian clubs, dumb-bells, both of very light weight at first, and with other house apparatus, artificial boat-rowing, horse-back riding, etc.

Warm baths are always beneficial, and the baths at Nauheim have a special reputation in Europe in this regard. But the same benefits may be derived under intelligent supervision at some of the warm baths in our own country, as at Hot Springs in Virginia and in Arkansas. The bath should be warm at first but may be cold later, to get the stimulating effect of cold through the skin upon the nervous system and the circulation. These effects are thus described by von Hösslin:

After a full bath of 16.5° C. (61° F.) of fifteen minutes' duration, the blood pressure increases, according to Oertel, from 125 mm. to 137 mm. After the lapse of three hours the blood pressure is still 7 mm. higher than before the bath. Oertel explains the increase of pressure as due to increase of tension and diminution in the diameter of the arteries. Increase of blood pressure is also observed after a cold douche. Blagowetschensky and Winternitz observed a similar effect after a rain douche, at 10° C. (50° F.) of fifteen to twenty minutes' duration. So also an ice-bladder applied over the region of the heart elevates the blood pressure by increasing the tone of the heart muscle and the vessels. Thus there occurs, after this procedure, an increase in the general tone of the vessels and in the peripheric circulation. According to Schweinberg and Pollack, the cold sitz bath brings about a considerable increase of pressure, and the pulse curve, when one is in the cold sitz bath, shows all the characteristics of a vessel with high tone and increased pressure.

This increase of pressure is effected through the nervous system, as Röhrig observed that after section of the vagi thermic and sensitive irritants had no effect upon the heart muscle. The influence of these agents is therefore reflex. Warm baths have the opposite effect. The dilatation of all the vessels of the skin in the hot full bath or in a steam bath reduces the blood pressure, unless this effect is counteracted by an increase in the tone of the vessels or in the energy of the contractions of the heart (Leichtenstern).

The best results in the use of baths are obtained, as already remarked in the study of hypertrophy, in institutions where the benefits of warm baths and mineral waters may be secured under exact supervision.

In the treatment of fatty degeneration of the heart the question will always arise whether exercise or rest is the better treatment for the individual case. This question may be met, in a general way,

with the answer that during the stage of defective compensation rest is indicated, and with the restoration of compensation the heart muscle may be better toned by graded exercise, especially in the open air. Later on, the patient may betake himself to outdoor exercise and may commence by climbing gentle acclivities, first stopping frequently to rest. Many, perhaps all, cases, at the start, may be best treated in this way without any appeal to drugs at all. In order that the exercise may be rightly done and not overdone, Oertel marks off certain promenades with different colored signs, and fixes the distance by poles. The patient is then instructed to take the plane marked by a red or blue sign and to cover the distance embraced between a certain number of posts.

But it is exactly in cases of fatty degeneration of the heart that the most extreme caution must be used with both diet and exercise. Schott (Nauheim) establishes the following principles: Reduction cures must always be undertaken with caution and should be limited to young people of normal nutrition and vigorous musculature. Any too rapid or too extreme reduction in weight is to be avoided. General diseases, indications of senility, etc., furnish, at least in the beginning, contra-indications to any reduction cure. The fatty heart may often be favorably treated without any reduction of weight. The dietetic mechanical treatment deserves preference over all other therapeutic measures.

Thus Roemer reports, out of 234 cases of heart weakness without valve lesion, 81 that were treated in this way without medication; of these cases 46 recovered permanently, 17 temporarily, while in 18 cases the treatment proved insufficient.

Where the patient is not benefited by rest in bed, with regulation of the diet, and later by properly graded exercise, or where the signs of heart failure are ominous, appeal must be had to digitalis, to which the heart will respond according to its capacity, that is, according to the amount of tissue which remains unaffected; for digitalis has, of course, no effect upon muscular structure in a state of fatty degeneration. But, as elsewhere observed, digitalis slows the action of the heart by its influence upon the vagus nerve, and in this way gives the heart muscle still unaffected time to recover tone. But digitalis is cumulative and should be administered continuously for but a short time, a week or two at most. The remedy should then be discontinued absolutely for a week or more, and may be resumed on the reappearance of any threatening signs. In this way life may be prolonged for years.

As mentioned under Prognosis, where digitalis fails the outlook is bad; nevertheless exceptional cases respond to other heart tonics,

strophanthus, spartein, etc. In the face of imminent danger of collapse resort is had to caffeine, camphor, ether, musk, after the manner specified in the treatment of chronic valve lesions. Liebermeister reports that, of 172 cases of heart failure without valve lesion treated by digitalis, including 21 cases previously treated on the expectant method without any or with but temporary effect, permanent restoration was secured under treatment by digitalis in 106, temporary results in 37, while in 29 cases the remedy remained ineffective.

Strychnine is the best remedy to sustain the good effects thus temporarily obtained by digitalis. The strychnine may be given in the form of a solution, especially the nitrate, one grain to the ounce, in a beginning dose of five drops three times a day, to be gradually increased to twice this amount. Or the pure solution may be substituted by the tincture of nux vomica in dose of ten to twenty drops three times a day. It is needless to state that a patient restored in this way may preserve the heart only by constant vigilance. Any imprudence is likely to be quickly punished, and, as a rule, out of all proportion to the indulgence. It is, therefore, wise to remember that in these cases "conservation is as hard as creation."

Details of treatment are further described in the section on myocarditis, hypertrophy, and chronic valvular disease.

Rupture of the Heart.

Rupture of the heart is of two kinds, traumatic and spontaneous. Wounds of the heart chiefly concern the right ventricle which, being apposed to the wall of the thorax, lies nearest to the surface of the body. Traumatic rupture belongs to the domain of surgery.

Spontaneous rupture was first observed by Harvey, and the fact that such rupture may occur only in a diseased heart was recognized as long ago as by Morgagni. But not at all infrequently the disease is latent and is unrecognized, and the rupture which terminates life suddenly and in the midst of apparent health may occur under circumstances of juridical importance. Thus Battalia reported a case of spontaneous rupture of the ventricle independent of any inflammation or of any external lesion; Beer a rupture of the heart without preceding demonstrable disease of the heart; Ward a rupture from external violence without breach of the skin; Gouzy a rupture in consequence of an occult carditis, and Fischer a rupture of an apparently sound heart. Tamburini reported a rupture of a fatty heart in consequence of a blow in a case which had medico-legal interest, and

Cocheteux a case of red softening with rupture and instantaneous death in which there was an accusation of parricide.

Rupture of the heart is found most frequently in the left ventricle. It is rare in the right ventricle, and still rarer in the auricles; but, when occurring in these places, is found rather more frequently in the right than the left auricle. Thus, according to the statistics of Elléaume, the rupture was found in the left ventricle 43 times, in the right ventricle 7 times, in the right auricle 3 times, in the left auricle twice. Similar results are shown in the later statistics of Ollivier, Pigeaux, Bertherand, and Quain. Among the recent cases Davis and Leaming reported rupture of the right ventricle; Lankester, Thomas, and Thompson each rupture of the right auricle; da Costa a sudden death from rupture of the right auricle by an acephalocyst; Clapton a rupture of the left auricle; Ashburner and Blauvelt rupture of both ventricles; Desmarets rupture of both auricles; Peacock and Höring rupture of the septum; Carter and Draper rupture of the septum from external violence; Beith rupture of the septum ventriculorum extending through the walls of the right ventricle; Hewett traumatic rupture of the ventricular septum without implication of the pericardium; Dransart rupture of the heart without involving the endocardium, and Maclagan sudden death from rupture of the superficial fibres of the heart.

According to Devergne, in every 40 cases of sudden death there is 1 by rupture of the heart. Aran found the proportion as high as 33 in 202 cases of sudden death occurring in the course of heart disease. The greatest number of cases occur in advanced life, after sixty. Men are affected more frequently than women, in the proportion, according to Elléaume, of 37 to 24.

Morbid Anatomy.—On opening the sac of the pericardium it is seen that the cavity contains blood. The amount which it may contain varies. Sometimes there are found only ecchymoses beneath the visceral layer of the pericardium. Sometimes the blood is coagulated in a thin layer or in several layers over the surface of the heart. Where there are several layers, the outside layers, being the most recent, consist of soft, loose coagula, while the inner layers are more white, firm, and adherent. The writer once stripped off five layers of successive coagula in a case in which the fatal termination had been delayed for several days after the rupture. The quantity is rarely so great as actually to distend the pericardial sac. Merklen recorded an exceptional case in which there was no blood in the pericardium.

On turning out the fluid blood and loose coagula, and stripping off the adherent layers, the line of the rupture presents itself at once.

Sometimes blood continues to ooze from the open wound. The rupture is found, as stated, most frequently in the left ventricle near the apex, and usually on the anterior face of the heart. Rupture of the posterior wall or at the base of the heart is rare, and still more rare is rupture of the septum.

Ruptures are said to be complete or incomplete according as they extend partly or wholly through the wall of the heart. The line of rupture is seldom straight; it presents a zigzag course, and the penetration is irregular so that the inside and outside openings do not exactly correspond. It is plain to see in all cases that the rupture extends from within outward under the force of the pressure of blood in the systole. Sometimes the rupture is limited to a small part of the heart wall, including for instance one or several trabeculæ, or, as already remarked in the study of lesions of the valves, the rupture may be limited to the papillary muscle. By rupture of the heart is generally meant, however, a rupture of the wall of the heart. The length of the rupture varies. It seldom measures more than half an inch; sometimes the orifice will permit only the passage of a sound. In rare cases the rupture may split the heart from the apex to the base. Rupture is usually single; sometimes it is multiple. Wiltshire reported spontaneous rupture in two places; Switzer a similar case; Packard rupture at three points with other lesions caused by a railroad accident; Schmucker multiple rupture of a thoroughly softened heart; Baly three ruptures, and Ollivier and Andral recorded five distinct ruptures in the left ventricle.

The following cases illustrate the cause of the condition and the nature of the accident. Mollière reported a case of sudden death from spontaneous rupture in consequence of thrombosis of the coronary artery with inundation of the pericardium; Spiegelberg a sudden death on the third day of the puerperium from rupture of the left ventricle in consequence of acute myocarditis; Shearer a case of sudden death from rupture of the right auricle in which there were small saccular aneurysms of the ascending aorta with hypertrophy and fatty degeneration of the heart; Wilkinson an aneurysm of the aorta with rupture of the sac, hemorrhage into the pericardium, and instant death in a case of inflammatory softening and rupture of the heart; R. W. Smith a fatty heart, with rupture of the left ventricle and the presence of free oil in the blood; Feldhahn the rupture of a thoroughly softened heart; and finally, as illustrative of the suddenness of death, Keen reported a rupture of the heart with post-mortem muscular spasms.

Etiology.—Rupture of the heart implies disease of the heart muscle, and the disease most frequently encountered is fatty degeneration.

In 62 cases of rupture Quain found fatty degeneration 25 times. Barth found fatty degeneration 19 times in 24 cases. The other changes of myocarditis may likewise lead to rupture; thus an abscess in the walls of the heart may break through into the pericardium or into the cavity of the ventricles. Regions of hyaline or amyloid degeneration may yield in this way, and aneurysm in the walls of the heart may rupture into the chambers of the heart or into the pericardial sac. Monneret found rupture 3 times in 19 cases of aneurysm of the heart (Schroetter).

Behind the degenerative changes may usually be found the lesions of arteriosclerosis. As might be inferred from the site of the rupture, the changes are usually encountered in the branches of the left coronary artery. Hertz, Mackenzie, Leyden, Trier, with a host of observers, report cases of atheromatous degeneration and thrombotic occlusion of branches of the coronary artery. The anæmic necrosis of the heart wall may likewise be referred to diseases in the coronary arteries. Robin laid stress upon the fragmentation of muscle fibre described by Renaut as a chief anatomical condition in rupture of the heart (Petit). Thus it may be understood that while the final event, the rupture, is sudden, the lesions which lead up to it are usually insidious and slow in development.

The immediate cause of the rupture is often a strain. The greatest number of cases occur after lifting a weight, carrying a burden, or climbing a hill. Finnel reported a case of fatty heart with rupture from fright; Maffei sudden death from extensive rupture in consequence of great fright; Hudson a case of rupture of the right auricle in labor; Duclaux death from rupture in spontaneous tetanus. Tissot recorded a case of rupture during an attack of epilepsy. In the case of George II., King of England, the rupture occurred during straining at stool. The first case of death during copulation, recorded by Morgagni, has since been multiplied many times by other observers. Rupture sometimes takes place in the act of vomiting, coughing, even of laughing. Not infrequently the heart literally breaks under the emotions of joy, anger, or fright. On the other hand, the accident occurs at times during perfect tranquillity of mind and body, as for instance during sleep.

Stolper reported a case of a workman, aged sixty-three, affected with emphysema of the lungs and bronchitis, which were improving under treatment in the hospital, who fell dead on the morning of his release while he was washing himself. There had been no particular strain. Post-mortem examination showed the pericardium distended with fresh blood clot, which enveloped the heart like a mantle and was fixed at the base. On the posterior surface at the apex of the

left ventricule, that is, at the point of predilection, there was found an irregular zigzag rupture, 2 cm. long at a point where there was a protrusion, the size of a walnut, of the venticular wall which was extremely thin. The cause was a myomalacia in consequence of sclerosis of the coronary arteries. Zurel reported a case of progressive rupture of the heart.

Beadles reported six cases of rupture of the heart among the insane. In every case the rupture was found in the left ventricle in connection with extreme atrophy of the muscle substance and fatty deposits. The valves were sclerotic and the kidneys cirrhotic. In most of the cases the physical condition gave rise to no suspicion of heart disease up to the moment of sudden death.

Moore found a rupture of healthy muscle in the left ventricle near the apex in the case of a boy, aged eight years, who had died of drowning. The pressure which had resulted from suffocation was regarded as the cause of the rupture.

Symptoms.—Rupture of the heart is usually announced by signs so severe as to be almost tragic. The patient is transfixed with a sudden stab of pain and falls into fatal collapse. Death is sometimes almost instantaneous. The patient is heard to cry out or seen to seize himself in the region of the heart, and falls to the ground as if stricken with apoplexy. More frequently, however, several hours elapse before the fatal termination. During this time the pain subsides to some extent, and the patient may become conscious; the body is covered with a cold sweat, and there are dyspnœa and cyanosis. Sometimes there are convulsions and not infrequently the patient falls into coma. During all this time the action of the heart is tumultuous and bounding. The patient may remain in this state for several hours or days, during which there are remissions and exacerbations, any one of which may terminate fatally. Protracted cases are marked by more distinct recovery, so that the patient may even go about for a time and remain comparatively free of symptoms, or be affected from time to time with attacks of nausea, vomiting, anxiety, vertigo, and syncope. During this period the patient may be free from pain or may suffer the excruciating anguish of angina pectoris. These protracted cases are explained by the fact that the rupture is but slight, or, when more extensive, is closed by a thrombus; and the symptoms are renewed or life is taken by extension of the rupture or by discharge of the clot. The symptoms and lesions are so well illustrated in the case of the distinguished physician, Professor Panum, as reported by Trier and recorded by Fraentzel, as to justify repetition here.

Professor Panum had reached the age of sixty-four years and four

months in fairly good health, though he had suffered for ten years from chronic bronchitis and some emphysema, which brought on a slight degree of dyspnœa in climbing stairs. It was only, however, in the last weeks of his life that he had to stop on the stairs in this effort because of a feeling of tension in the chest. This feeling disappeared with a few minutes' rest. One evening, on returning home about seven o'clock, he noticed that he was unable to keep up with his companion in the face of a strong head-wind, but was compelled to stop here and there to recover his strength. On reaching home he was seized suddenly with pain in the region of the heart. It was a feeling as if something gave way in the left side of the chest. The pain continued to increase and was associated with anxiety. Trier found his patient at ten o'clock pale, prostrated, with cool extremities, small, frequent, and irregular pulse; but the speech was clear and respiration was but little hastened. The patient moved about his room, undressed himself rapidly and went to bed. The pains now radiated from the sternum to the left arm and down to the fingers, and were so severe as to require the utmost effort to conceal the suffering. The remedies administered had no effect as they excited nausea and vomiting, but the sensorium still remained perfectly clear and the patient in a half-jesting way ridiculed the failures to secure relief. But sinapisms, hot applications, stimulants, and a large injection of morphine remained without effect. It was only in the course of half an hour after the injection that the pains began to give way and there was an inclination to sleep. The sleep was short, restless, and interrupted, but vomiting did not recur until the next morning after taking coffee. The patient felt now so little sick as to be persuaded with difficulty to remain in bed. He explained his illness to his son as due to a nervous disturbance of the vagus and sympathetic, but declared that he could not work out the cause. The appearance had now improved and the pulse had become strong. The son left him for half an hour, and in the course of a few minutes, while he was jesting with some one who had just stepped out of the room, he was heard to utter a cry and was found cyanotic and comatose. Death ensued in a few minutes. It should be said that the superficial examination made on the previous evening had demonstrated no increase in dulness, but that there was a tumultuous action of the heart and that the presence of a loud, long-drawn, blowing murmur made it absolutely impossible to distinguish the two heart sounds.

The post-mortem revealed a considerable accumulation of fat in the mediastinum which covered the whole anterior surface of the pericardium. The upper lobes of the lungs on both sides and the

middle lobe on the right side were found emphysematous, especially the anterior borders. On opening the pericardium the sac was found filled with dark, partly fluid, partly coagulated blood. On withdrawing the heart, blood of similar consistence escaped from a rupture on the anterior surface of the left ventricle. The split ran in an almost straight line, parallel with and very near to the septum. The edges were notched and torn. A smaller similar rupture ran parallel with the larger rupture and was connected with it by a transverse line. The larger rupture was 5, the smaller 1½ cm. long. They both extended through the thick layer of blood-infiltrated fat under the pericardium into the muscle substance, which was penetrated by several small ruptures, with openings so small as to permit only the passage of a fine sound. At the end of the rupture the sound passed into a small cavity which lay between the layer of fat and the musculature and was filled with dark fluid blood. The heart was lax and covered with fat in a layer at the apex over 1 cm. thick. There was abundant evidence of atheroma of the aorta. The vertical branch of the coronary artery was so contracted as to admit only the passage of a very fine sound. At this region there was a soft thrombus covering a small atheromatous cavity in the calcified intima, which was penetrated by numerous small openings.

Microscopical examination of the heart muscle showed the fibres in the region of the rupture atrophied and in a state of extreme fatty degeneration. All striation had disappeared. In several regions the muscle fibres were torn and separated by extravasated blood.

The sudden death and the symptoms which had preceded it met a satisfactory explanation in the rupture of the heart with the escape of the average quantity of blood into the pericardial sac. The rupture depended upon fatty degeneration and atrophy in the circumscribed portion of the anterior wall of the left ventricle, and here could be demonstrated atheromatous degeneration of the branch of the coronary artery with a thrombus which almost blocked the lumen of the vessel. It was supposed that the thrombus and the subsequent degeneration and atrophy had developed in the course of the last weeks of life, and had given rise to the increased dyspnœa and tension in the chest upon exercise. The moment of rupture was felt by the patient himself as the cause of the extreme pain and tumultuous action of the heart with the subsequent nausea and vomiting. As the rupture did not immediately traverse the entire thickness of the wall there was at first no blood in the pericardium and no increase of dulness.

The sudden death which occurs in these cases cannot be explained by interference with the action of the heart in consequence of the extravasated blood, for the heart may still continue to do its work in

the face of the much greater pressure of a pericardial effusion; nor can it be attributed to anæmia of the brain, for many cases show no signs of anæmia; it is best explained by shock and sudden paralysis.

The *diagnosis* is difficult, as a rule, and is often impossible. Where the rupture occurs as a sudden catastrophe in a patient in advanced life, known to have been affected with heart disease, especially with fatty degeneration or myocarditis, and shows itself as a seizure of violent pain followed by rapid collapse, with tumultuous action of the heart, dyspnœa, cyanosis, convulsions, and coma, the diagnosis of rupture may be accepted. In such circumstances the condition would require to be separated only from angina pectoris. The history of previous attacks of pain of anginose character may here make the distinction. The pains which occur in rupture are to be attributed to the thrombotic occlusion of the coronary arteries which cause the angina. Cases of rupture of long duration are distinguished by exacerbation and remission of symptoms in the course of several days. Angina pectoris occurs, as a rule, in attacks separated by much longer intervals. Where the course of the affection is more protracted, the escape of blood into the pericardial sac may be recognized by the increase of dulness.

The *prognosis* is bad. The rupture itself often takes life at once. The condition behind it is usually irremediable. The duration of life varies, as a rule, from a few hours to a few days. Sprakeling reported a rupture from violence with death after eight hours; Stroud a case of spontaneous rupture of the heart in which life continued for about ten hours; Fenner a case in which the patient survived twenty-eight and a half hours; Coupland a spontaneous rupture with survival for forty hours. In the case mentioned in the experience of the writer, the patient lived four days unconscious, comatose, and cyanotic during the whole period. Peter mentions a case which survived twelve days. Even cases of recovery have been recorded. Thus, Meyer reported a case of rupture of the left ventricle terminating in recovery. Beadles records a case which survived for one hundred and sixty-eight days, probably on account of temporary closure of the wound by a thrombus. Rostan saw a rupture closed by clots of fibrin, and speaks of a closure under agglutination of the pericardium which remained perfect for fifteen years, at the end of which time death occurred in consequence of a new rupture.

Treatment.—There is not much room for treatment, but where the case is at all protracted effort may be made to arrest the hemorrhage, especially by the application of cold in the form of the ice-bag over the region of the heart, the action of which is quieted as much as possible by absolute rest. Venesection, with a view of checking

the hemorrhage by weakening the heart, is not to be considered for a moment. The tumultuous action of the heart does not depend upon weakness, but upon disturbance in the nervous mechanism, and this disturbance is better quieted by the application of cold. The effort may be made to derive the blood from the heart to the extremities by sinapisms and hot baths. Friedreich suggests the possibility of relief by the application to the lower extremities of the Junot boot, which dilates and fills the vessels by exhausting the air from the surface. The pains of angina pectoris should be relieved by the subcutaneous injection of morphine in sufficient dose, and by the inhalation of amyl nitrite. In the face of profound prostration and pallor, with feeble and fluttering pulse and almost imperceptible action of the heart, resort should be had to stimulants, such as alcohol, ether, camphor, caffeine, and digitalis.

Syphilis of the Heart.

Intimations of syphilis of the heart may be found in the writings of the older authors. Corvisart considered as syphilitic excrescences or condylomata the vegetations or verrucosities which are since known to be the results of endocarditis. Morgagni certainly saw syphilis of the heart, and Ricord reported cases. But the true literature of syphilis of the myocardium begins with Virchow (1859), who recognized the evidence of constitutional syphilis in gummata of the heart muscle. Since this time a number of cases have been recorded by Cantani, Gould, Lancereaux, Marchiafava, Schwalbe, and Pitt, and the subject was made a special study by Mracek (1892). This author collected from the literature 102 cases, in 61 of which the nature of the affection was established by autopsy, and 10 of which actually occurred under his own observation. Syphilitic affection of the heart is not frequent, but recorded cases multiply rapidly under modern observation.

Muscular tissue is not selected by syphilis, and though the evidences of syphilis are sometimes found in the muscle, the heart itself is least frequently affected. Hereditary syphilis almost never attacks the heart, though cases of this kind were reported by Parrot, Wendt, and Foerster, and the signs of constitutional syphilis are found only in the latest periods of the disease. Consequently syphilis shows itself in its tertiary manifestations in the heart, and, as a rule, not sooner than eight to ten years after the period of primary infection.

With other localizations, heart syphilis occurs more frequently in the male sex. Of 31 cases only 10 were found in women. Most of the cases occur between the ages of twenty and forty.

Syphilis may attack any part of the heart—the muscle of the heart, the membranes, and the blood-vessels. The common manifestation is the gumma. But syphilis also produces a particular myocarditis. The cases of Mracek were as follows: Myocarditis gummosa, 10; myocarditis fibrosa, 9; myocarditis gummosa et fibrosa, 8; endocarditis gummosa, 2; pericarditis gummosa, 1; disease of vessels 3; myocarditis with peri- or endocarditis, 15; peri- and endocarditis, 1; disease of vessels of the myocardium, 1; disease of all organic parts, 6; rhabdomyoma, 1; disease of the ganglia, 4. As is seen in this statement, various lesions may coincide.

The heart gummata vary in size from a pea to a pigeon's egg. They are sometimes embedded deep in the muscular tissue or may protrude to form a tumor upon the surface. They are most commonly found in the ventricles, especially the left ventricle, but are encountered also in the auricles, in the septum, and in the papillary muscles. The largest tumors are found in the septum, where they may form a mass which protrudes into both cavities. Gummata which approach the endo- or pericardium excite inflammation in these structures. They are usually multiple and present the pale gray appearance and peculiar consistence, characteristic of the deposits elsewhere. They may remain encysted or may subsequently break down under a process of softening, to form abscesses or to discharge their contents into the sac of the pericardium or cavity of the heart. Emptying into the cavity of the ventricles they may form emboli in distant organs.

The syphilitic sclerotic myocarditis represents a cicatricial degeneration after necrosis from obliterating endarteritis. Affected organs show the characteristic fibroid indurations, with atrophy of the muscular and substitution by cicatricial tissue, which are sometimes interpenetrated by semi-gummatous nodules (Lancereaux). Partial aneurysm of the heart was found in eight of the cases recorded by Mracek. The disease may be circumscribed, but is usually more or less disseminated and is often associated with endo- and pericarditis. Foerster described a syphilitic endocarditis confined to the mitral and tricuspid valves in the case of new-born children affected with hereditary syphilis. Similar localizations have been recorded in the chapter on sclerotic endocarditis.

Syphilitic endocarditis may be parietal or valvular, but a large number of cases recorded as syphilitic endocarditis are really secondary affections during the second stage of syphilis (Mracek).

It has already been remarked that valvular deformities may ensue in consequence of the retraction of such cicatricial tissue in the papillary muscles. Dehio claimed that no case had been reported

of insufficiency or stenosis from adhesion or shrinkage of valves due to syphilis of the endocardium, but such cases may nevertheless be found in the literature. Thus Leared described a case of syphilitic affection of the aortic valves, and Fraentzel reported a case of an old man who had been treated for insufficiency of the aortic valve for three years, and who showed the signs two weeks before death of insufficiency of the tricuspid valve. The autopsy showed adhesion of the left anterior tricuspid valve to an endocarditic neoplasm, which Cohnheim recognized as a metamorphosed gumma. Schwalbe described in Virchow's *Archiv* a case of syphilitic stenosis of the pulmonary artery with insufficiency of the pulmonary valves.

The syphilitic endarteritis is usually the initial lesion, and more or less evidence of syphilitic degeneration of the coronary arteries may be disclosed in every form of heart syphilis.

The pathogenic influence of syphilis in the production of aneurysm has long been known. Welsh maintained that syphilis was the cause of true aneurysm in sixty per cent. of cases. Other authors put the percentage as high as eighty. Syphilitic affections of the coronary artery have been frequently reported, as by Birch-Hirschfeld, Chvostek, Weichselbaum, Ehrich, and Fraenkel. The case reported by the first of these observers was interesting in the fact that the patient, a man aged twenty-five, had suffered for four years, during the existence of syphilis, with frequent attacks of palpitation of the heart and angina pectoris. The post mortem examination showed the signs of liver syphilis. In the case of Fraenkel the patient had suffered from headache with occasional attacks of syncope and violent attacks of angina pectoris. The case was that of a woman whose husband had been syphilitic and who had herself suffered formerly with tumors of the head that had discharged and left cicatrices.

At the post-mortem the condition of the coronary artery was especially studied. The anterior or left coronary artery was perfectly patulous and enlarged, while the posterior or right coronary artery was totally obliterated at its orifice in the aorta in consequence of an advanced arteriosclerotic process. The trunk of the vessel and the position of the obliterated orifice could be demonstrated in the sulcus ventricularis only after dissection and careful penetration by a sound. That these changes were really syphilitic was shown by the existence of a gummatous tumor in the septum ventriculorum, having a diameter of 4½ cm. The aorta showed arteriosclerotic change in its entire extent down to the iliac arteries.

Finally, syphilis affects the nerves of the heart. Schott found syphilis to be the sole cause of neurosis of the heart in three cases.

Saccharjin once cured a case of syphilitic cardiac asthma by specific treatment.

Symptoms.—Syphilis of the heart may show no signs, as the course of heart syphilis is slow and insidious. A gumma may remain a long time encapsulated without symptoms. The signs which show themselves do not differ from those dependent upon myocarditis from other cause.

Syphilitic myocarditis is an important factor in producing the weakness and cachexia of the later stages of the disease. Most cases show, sooner or later, early exhaustion upon effort, panting respiration, dyspnœa, venous stasis, œdema about the ankles, and dropsy. The disease is often so insidious as to show signs only when the heart muscle has been extensively involved. Hence it is that sudden death is frequent, and much more frequent than it would be if the nature of the affection were recognized earlier.

The *diagnosis* rests upon the fact that the signs of syphilis may be found elsewhere. It is the occurrence of the evidence of myocarditis in the syphilitic subject which makes the diagnosis possible. Syphilis leaves traces which may always be discovered under scrutiny. Thus sometimes osteophytes are felt upon the legs, or faint cicatrices or pigmentations about the tibia stamp the character of the disease. Enlarged lymph glands may be felt in the sub-occipital and post-cubital regions. Sometimes there is evidence of alopecia. Patients complain of pain, sometimes fixed and deep-seated, in the bones, especially in the bones of the skull, tibia, and ribs. The signs of dilatation of the heart are sufficiently pronounced, and the cause of the condition is revealed so soon as it is suspected. Any doubt is soon dispelled by the efficacy of antisyphilitic treatment. One of the most satisfactory cases which the writer of this article ever had was that of a fashionable lady at a watering-place, who suffered attacks of vertigo and dyspnœa, at first only upon exercise as in the ball-room, later upon lighter provocation, as after the ingestion of strong coffee or emotional disturbance, and who showed distinctive evidence of dilatation of the heart in a weak and irregular pulse and œdema of the feet, in association with physical signs on the part of the heart itself. This patient had wandered wide in a vain search for relief. The case had been properly diagnosticated as a myocarditis, and digitalis and other heart stimulants had been regularly administered, and always without any real avail. The request for a remedy for falling of the hair first excited the suspicion of a possible syphilis, and a slight examination into the history soon disclosed the previous existence, many years before, of an eruption, sore throat, ulcers of the tibia, one of which in fact existed at the

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time, with other unmistakable signs of the disease. Under the inunction treatment this patient made a rapid recovery.

The *prognosis*, as a rule, is bad, but will not remain so unqualifiedly bad as hitherto given because of the possibility in the future of earlier recognition. The prognosis depends upon the extent of the disease, the general cachexia, and the gravity of the heart symptoms. When it shall come to pass that the nature of the affection will be made out before destructive changes occur, the prognosis will become more favorable. At the present time most cases succumb, either suddenly to heart failure or gradually under progressive marasmus. It will be remembered that, as in the case of the more remote lesions of syphilis elsewhere, though the deposits (gummata) may be made to disappear, irreparable damage may be left behind. The prognosis, therefore, takes on the gloomy aspect of the post-syphilitic phenomena of tabes dorsalis, progressive paresis, etc. As is observed so often in the brain and cord, gummata may be cleared away, but interstitial changes may not be eradicated. The main hope of treatment hinges upon early diagnosis with the institution of treatment before there is time for destruction of tissue and other irremediable change. When the specific treatment of syphilis shall be continued regularly or interruptedly for years, cases of heart syphilis will become still more rare.

Treatment.—The treatment consists in the administration of mercury and the iodides. Mercury furnishes the best results by inunction, and every patient affected with heart syphilis should be put at once upon the inunction cure. In fact, the mere suspicion of syphilis justifies the treatment. The treatment may be interrupted from time to time, after the lapse, for instance, of several weeks or months, and substituted for a time by the potassium or sodium iodide, which may be given in the dose of grs. v.–xv. three times a day, best largely diluted in milk. The iodine treatment should be itself suspended in the course of several weeks and the mercurial inunctions resumed.

It is needless to state that the signs of heart failure must be combated by the usual means, especially by rest, by the exhibition of digitalis, and by general tonics, iron, quinine, and strychnine.

Aneurysm of the Heart.

Under the term aneurysm was formerly understood the enlargement of the heart caused by hypertrophy and dilatation. Both Avenbrugger and Corvisart considered these conditions as aneurysma cordis, and distinguished the hypertrophy and dilatations as

aneurysma passivum and activum. It was in this sense that Cholmeley described a case of fatty degeneration of the heart with adherent pericardium and membranous pouching of the left ventricle, that Portal speaks in his memoir of aneurysms of the heart in which the walls of the heart instead of being thin have preserved their natural thickness or have even increased in size and that Pollock reported an aneurysmal dilatation of the right auricle from contraction of the right auriculo-ventricular opening. The term is limited in our day to dilatation of part of the heart and is usually characterized as partial aneurysm. Avenbrugger, however, also noticed this condition, which was more especially remarked by Boerhaave and Van Swieten. Rokitansky enriched the subject with numerous observations, and Pelvet (1867) collected all the cases published up to his time. Since this period reports and demonstrations of cases have constantly been made.

Aneurysm of the heart is limited to the dilatation which takes place in the walls of the heart. Aneurysm of the valves is considered under the subject of valve lesions.

Aneurysms are distinguished as acute and chronic according to the rapidity of their formation. The acute aneurysm is the sudden dilatation of a weak spot in the myocardium under a reflux of blood. The weak spot represents a centre of myocarditis or of softening, a myomalacia, the result of occlusion of a branch of a coronary artery. Sometimes the process starts from the endocardium, which suffers necrosis at some point, to make of this region literally a *locus minoris resistentiæ*. The acute aneurysm is almost confined to the left ventricle, near the apex, and is located especially in the anterior wall. Occasional cases are also encountered in the upper portion of the ventricular septum. The acute aneurysm indicates rapid increase of size and usually takes the life of the patient in a short time by the discharge of its contents into the chambers of the heart with the production of embolism, or into the pericardium under the picture of rupture of the heart. McNalty reported an aneurysm at the apex of the left ventricle bursting into the pericardium and causing immediate death.

Chronic aneurysm is nearly always a result of a localized myocarditis. The influence of syphilis in this regard has already been remarked. As mentioned elsewhere, Welsh maintained that syphilis was the cause of true aneurysm in sixty per cent. of cases. Other authors put the percentage as high as eighty. Francis encountered a case of aneurysm of the heart with syphilitic deposit, Pitt the case of a young man, aged twenty-eight, who died during antisyphilitic treatment, in whom there was found in the left ventricle an aneurysm

which had burst into the pericardium. The wall of both sides of the heart showed extensive gummatous changes. There was sclerosis of the aorta, and also cicatricial degeneration of the testicle. Other cases may be accounted for by atheroma of branches of coronary arteries. The cicatricial connective tissue which represents the fibroid degeneration yields under the pressure of the blood, so that a cavity is gradually formed which communicates with the chambers of the heart. The seat of this aneurysm is the same as that of the acute form, that is, the cavity forms in the wall of the left ventricle and, as a rule, near the apex. Pelvet found the aneurysm near the apex of the left heart in eighty-five of eighty-seven cases. When the aneurysm involves the septum it bulges always into the cavity of the right ventricle. Recent cases of aneurysm of the left ventricle are reported by Landouzy, Canton, and Bellingham. In the case of Lacanal an aneurysm the size of an egg protruded at the apex of the left ventricle. In the cases of Cruveilhier and Cunier the aneurysm of the left ventricle was attended with calcification of the aorta. Arnott reported a case of aneurysm of the left ventricle of the heart with partially ossified walls winding around the root of the aorta. In the case of Townsend the aneurysm of the left ventricle was itself in a state of calcification. Marie reported a case of a man, aged seventy-three, in whom was found on autopsy an aneurysm the size of a hen's egg on the front wall of the left ventricle near the apex. The walls of the aneurysm were completely calcified so that they had to be cut through with a saw. There was extensive sclerosis and atheroma, especially of the left coronary artery, which was looked upon as the cause of the aneurysm. In the case of Fredet aneurysm was found in connection with calcareous degeneration of the walls of the heart. Janeway, Pozzi, and Tournilhac-Beringier each reported cases of double aneurysm of the heart, Little a case of bilocular aneurysm of the left ventricle. Legg found an aneurysm of the right auricle, Legroux an aneurysm of the left auricle; Leudet a partial aneurysm of the left auricle, with a voluminous clot in the left ventricle. La Sauvage saw a case of apoplexy, the result of aneurysm of both auricles of the heart. Wiegandt found an aneurysm of the septum of the heart. Zahn reported two cases of aneurysm of the septum of the ventricle, Griesinger an aneurysm of the ventricular septum with communication between the two ventricles, and one of the right ventricle communicating with the orifice of the aorta. Legg saw an aneurysm of the septum of the heart in a case of phthisis with contracted kidneys. There was general extreme atheroma with aneurysm of the left internal iliac artery, and thrombosis of the inferior vena cava and both iliac veins. Irvine saw a defective muscular development of the car-

diac ventricular septum with consequent aneurysm of the septum and displacement of an aortic valve by the aneurysm. Finally Brichetau reported a case of aneurysm of all the cavities of the heart in a young girl aged twelve years.

The aneurysmal tumor varies in size from a scarcely recognizable sac to a mass as big as half an apple (Kundrat) or orange (Ormerod). In Ormerod's case a laborer, aged thirty-five, died suddenly from unknown cause; the autopsy revealed an unruptured aneurysm the size of an orange in the upper part of the left ventricle lined with layers of clots; the cavities of the heart were slightly dilated; in other respects the heart, including the valves, was perfectly sound; the aorta was also sound. Petit speaks of very rare cases in which the aneurysmal sac was as large as the heart, so that the heart appeared to be double. Berthold reported a case in which an aneurysm distended the right auricle to the size of a man's head. There was universal contact in front with the walls of the chest, where a portion of the sternum and adjoining rib had been eroded, and the skin itself had suffered erosion to such degree as to permit the trickling of blood in various places. The aneurysm is usually single, but is sometimes multiple, and as many as four have been encountered, usually closely apposed to each other. The wall of the aneurysm is thinned by pressure, though it is often tough on account of its fibrous tissue. The cavity is filled with clots. Rupture is sometimes prevented by agglutination with the adjoining pericardium. Occasionally the walls of the aneurysm are calcified to a greater or less extent.

Symptoms.—Aneurysm of the heart presents no characteristic symptoms. The condition is commonly latent and is disclosed only upon autopsy. Not infrequently the end catastrophe, that is, the sudden irruption of emboli or rupture of the heart itself, will have been preceded by the evidence of weakened action. Arhythmia, intermittence, palpitation, dyspnœa, venous stasis, and œdema indicate the heart weakness of myocarditis from any cause.

The aneurysm is usually so small as to present no external evidence of its presence. In rare cases the sac has come in contact with the wall of the thorax, which it may erode, as in the example cited. Skoda also reported a case of visible tumor in the front wall of the thorax. Percussion would furnish dulness in only the most exceptional case. By auscultation Paul claims to be able to perceive a diastolic murmur at the apex due to reflux of blood from the aneurysmal sac into the ventricle at this time. Rendu remarks upon the existence of a diastolic metallic sound (*un bruit de claquement diastolique*), which he ascribes to tension of the sac. This bruit is more clear and sonorous than the metallic sound observed in renal cir-

rhosis. It occurs immediately after closure of the sigmoid valves and is heard in greatest intensity over the centre of the ventricle, whence it is propagated toward the ensiform cartilage. Bucquoy and Hanot place reliance upon a particular change of posture, in that subjects of aneurysm have a tendency, when seated, to lean forward until the forehead almost touches the knees (Petit). These various signs pertain really only to individual cases.

The *diagnosis* is, therefore, difficult and for the most part impossible. Suspicions may be entertained, but in only the rarest cases may the condition be recognized during life. Turnbull reported an aneurysm of the left ventricle with dropsy and physical signs resembling those of mitral insufficiency. The dropsy was removed by diuretics. The case terminated in sudden death.

The *prognosis* is not favorable. The tendency is toward continued expansion and final rupture. A somewhat better prognosis may be entertained in cases where the pathogenic relation of syphilis may be established by the evidence of signs of the disease elsewhere.

The *treatment* is wholly symptomatic. Wilks claims to have cured aneurysm of the heart, and Pouget speaks of treatment of an aneurysm of the heart by means of the moxa and the application of ice.

Thrombosis of the Heart.

Thrombi were first seen in the heart by Corvisart, who designated them as polypi and established the rules by which the ante-mortem are distinguished from the post-mortem clots. This distinction, said Laennec, is easy to make. The ante-mortem concretions are primary and more adherent to the walls of the heart, whose sinuses and intertrabecular spaces they penetrate. A recent concretion thus interlaced is still soft and may be detached in one mass. The older deposit is agglutinated and is torn in the act of separation. The older concretions are found most frequently in the sinus of the right auricle and in the right ventricle. They may completely fill the sinus of the auricle, and in the ventricle may double the thickness of its walls, contract the cavity and insinuate themselves under the tricuspid valve, whose action they embarrass. Moreover, the columnæ carneæ, to which they are adherent, are ordinarily much flattened, a condition which proves a development before death, for considerable time is required to produce this result. Corvisart in one case saw the fleshy columns effaced.

These rules remain in force up to the present time, though the designation of the forms has been substituted by terms which better express the different conditions. Since the time of Virchow the ante-

mortem deposit has been known as a thrombus, while the coagulation which takes place in the death agony or after death is known as the post-mortem clot.

True thrombi are then not simply loose deposits of blood or coagula which are insinuated between the trabeculæ. Thrombi adhere to the wall of the heart, change their color, assume a pale gray or violet hue, become drier, and may soften in their interior in the course of time to a puriform mass.

Hertz divides heart thrombi into four classes:

(1) The small accumulations adherent to the wall, softened in the centre so as to present the appearance of cysts. These are the globular vegetations of Laennec.

(2) Large masses which may fill a large section of the heart. Such masses are found especially in the appendices of the auricle.

(3) Pedunculated thrombi. These are the real polypi of the heart.

(4) Free masses nowhere adherent, floating as ball thrombi in the blood current.

In connection with a case of polypus of the heart under his own observation, Pawlowski collected the cases of the current century, 19 in all, of which 17 were tolerably accurately described. Of these cases there were 11 in women and 6 in men. It is remarkable that the septum of the heart, and especially the foramen ovale or its near vicinity, is the most frequent site of attachment of the heart thrombus, while the auricle is the seat of selection in the case of polypus.

The ball thrombi are found with especial frequency in stenosis of the mitral orifice as free bodies in the right auricle, driven about by the current of blood. Liebermeister saw a case of free thrombus in a valve lesion of this kind 2 cm. in diameter, and as elastic as a rubber ball. Henderson saw a globular polypus in the right auricle, and Legg two cases of loose balls of fibrin in the left auricle producing mitral stenosis.

The occlusion which may be produced in the heart by thrombosis is illustrated in the following cases:

Vernon saw obstruction to the passage of blood through the right auriculo-ventricular orifice from a fibrinous concretion which was entangled under the tendinous cords of the tricuspid valve; Caron a polypiform tumor in the left auricle extending into the left auriculo-ventricular orifice, which it blocked; Aran a case of organized thrombus almost completely obstructing the right ventricle and the orifice of the pulmonary artery; Richardson embolism of the right ventricle completely obliterating the calibre of the pulmonary artery and causing rapid death. Bellingham saw death caused by the for-

mation of fibrinous concretion in the right cavities of the heart obstructing the tricuspid orifice and extending into the divisions of the pulmonary artery. Schirwindt reported an interesting case of sudden death from the injection of a solution of sesquichloride of iron into a vascular tumor.

Etiology.—The coagulation of blood in the cavities of the heart is a consequence of disease of the blood or of the wall of the blood-vessel. Retardation of circulation contributes an important factor, but may not suffice of itself to produce a thrombus. The coagulation of blood in life is a process which depends upon the contact of fibrinogenous substance with fibrin ferment. These substances accumulate in quantity sufficient to produce coagulation only after dissolution of the leucocytes of the blood. Dissolution of the blood is brought about under the action of toxins of the infections. Ponfick showed that the endothelium undergoes fatty degeneration in the grave infections and is desquamated. The accumulation of these cells may start the process of coagulation. Every lesion of the wall of the vessel is marked by the accumulation of white blood corpuscles, which are subsequently converted into finely granulated fibrin. The dissolution of these corpuscles liberates the fibrin ferment which they contain, and the union of this ferment with the fibrinogenous element of the blood produces the thrombus. When the circulation is rapid, the thrombus is composed more or less exclusively of the white corpuscles; when it is retarded the red corpuscles are thrown from the centre to the circumference of the vessels and take part in the construction, constituting thus the red or mixed thrombus.

The development of the heart thrombus together with the process of thrombosis in other vessels occurs especially in diseases marked by poisoning of the blood, with retardation of the circulation and weak action of the heart. The heart thrombus is found, therefore, in marasmus from any cause, especially in the course of long-standing disease, tuberculosis, carcinoma, etc., in myocarditis and in the grave infections, especially in typhoid fever, on account of its protracted course and pronounced blood-poisoning. Tait has reported deaths from heart clot after abdominal operations. Playfair speaks of cardiac apnœa after delivery.

Symptoms.—The heart thrombus is usually latent and is disclosed for the first time at the autopsy. Small deposits may in no way interfere with the action of the heart, and even considerable thickening of the walls of a cavity may not materially diminish the blood supply under the weakened action of the heart. On the other hand, the symptoms may be sometimes severe. The gravest signs are furnished by the loose thrombus, as by such masses the natural orifices

may be closed or the action of the valves impeded. Again, masses may be detached and constitute emboli, which may block the branches of the pulmonary artery, or proceeding from the left heart may lead to occlusion of the cerebral vessels, infarction of the kidney, etc. Curious are the cases in which the thrombi penetrate from the right to the left auricle through an open foramen ovale, and lead to embolism in the systemic circulation. These are the paradoxic emboli of Zahn.

The *physical signs* are, as a rule, indistinct, but the sudden supervention of a bruit may indicate interference with the action of the valves. For the most part murmurs are absent because of the extremely weak action of the heart.

The vague and varied symptomatology is best illustrated by cases. Thus Oulmont reported a case of cyanosis and acute œdema, with a measly eruption, followed by death. The autopsy revealed cystic clots at the apex of the heart with deposits of pulmonary apoplexy. Baskin spoke of nausea and vomiting as symptoms of cardiac polypus. Lavirotte observed an exaggerated sonorousness of the lungs as a sign of the presence of clots in the right cavity of the heart. Fletcher saw an organized polypus of the heart, arising in the left auricle and hanging into the ventricle, which gave rise to a peculiar bruit distinctly audible some yards from the patient. Redtenbacher reported the case of a woman, aged forty-six, who showed the signs of a distinct mitral stenosis. The autopsy established the existence of a free-ball thrombus in the left auricle and also of a polypus dependent by a long pedicle into the auricle. The diagnosis was not made because of the absence of circumscribed gangrene in the feet, which had been considered by v. Ziemssen as pathognomonic. McGilliway reported fibrinous polypi in the right heart accompanied with tricuspid regurgitation in a case of double pneumonia. Pawlowski had under observation a female teacher, aged forty-seven, who showed the signs of stenosis of the left auriculo-ventricular orifice, though the presystolic murmur was often absent. The stenosis was caused by the presence of a polypus of the size of a walnut seated in the left auricle and attached to its posterior wall in the neighborhood of the orifice of the pulmonary veins. The polypus was composed of fine granular detritus with fat cells and pigment. Finally, Krumbholz reported two cases from the Leipsic clinic. In one, that of a seamstress aged forty-one, who showed the signs of mitral insufficiency and stenosis of the left auriculo-ventricular orifice, a free-ball thrombus of the form and size of a hen's egg was found in the left auricle. One pole showed a point which had been apparently in connection with the left auricle. The thrombus had a laminated construction and was covered with endo-

thelium. The diagnosis was not established in life because of the absence of infarction and gangrene. In the second case, that of a man, aged fifty-four, who had showed the signs of chronic peritonitis with ascites, there was found a pear-shaped polypus clinging with a pedicle, as thick as a lead pencil, to the side wall of the right auricle. The polypus reached down so as to extend into the right auriculo-ventricular orifice on one side and into the superior vena cava on the other. The inferior vena cava, shortly after its issue from the diaphragm, contained a flat mural thrombus. The heart polypus had an endothelial covering.

The *diagnosis* is difficult and usually impossible. The most stress may be laid upon the sudden occurrence of embolism. Krumm reported a curious case in which both lower extremities and the left upper extremity became gangrenous and no explanation of the occurrence could be found in life. The autopsy revealed as a cause of the condition a pedunculated fibroid polyp in the left apex developed from a cicatrix in the myocardium. From this mass peripheric emboli were detached to occlude the iliac and the left axillary artery. D'Espine reported multiple infarctions from clots imprisoned in the cavernous anfractuosities of the left ventricle.

Rosenbach heard in one case of mitral stenosis, with ball thrombus of the size of a nut in the left auricle, a loud presystolic and diastolic *frémissement cataire* in the second and third intercostal space near the left border of the sternum, while the thrill characteristic of the mitral stenosis could not be felt at the apex of the heart. The author intimates that this localization of the thrill may be of diagnostic value in the absence of any anomaly at the pulmonary orifice.

The remarkable irregularity of the symptomatology may excite the suspicion of the presence of a movable body in the cavities of the heart. Sometimes a diagnosis is reached. Malgaigne reported a case of extensive burn, with fever on the twelfth day and intermittence of the pulse; the diagnosis of fibrinous concretions in the heart was verified upon autopsy.

The *prognosis* is bad, not only on account of the mechanical interference with the action of the heart and the danger of embolism, but also on account of the underlying condition. The detachment of a large thrombus may block an orifice and cause sudden death by interruption of the circulation, or a large mass detached from the right heart may plug the trunk of a pulmonary artery and cause sudden death by suffocation. Fortunately, such a catastrophe is rare; more frequent is the separation of smaller masses with embolism in the different organs. At the same time it must be admitted that the condition is not necessarily fatal. Thrombi sometimes become

organized, and, as in the case of aneurysms, simply thicken the wall of the heart with an additional layer of connective tissue. Calcification sometimes occurs, or, as stated, softening (suppuration) with the additional danger of embolism. Thomson saw in the heart clots of blood organized or traversed by numerous vessels coming from the surface of the ventricles and auricles, and he injected these vessels from the bronchial arteries. In the case of Voelcker the thrombus was somewhat vascular and was covered by endothelium. There was no endocarditis and no infarction in the various organs. Ogle reported a case of fibrinous coagula of long standing which had undergone a process of softening and, in some cases, had become partially converted into a puriform fluid. Bristowe described softening clots in the heart.

The *treatment* is mainly addressed to the underlying condition. The weak action of the heart calls for the use of the analeptics and of digitalis, with caution, however, for overstimulation entails the danger of detachment of adherent thrombi.

Tumors of the Heart.

Tumors of the heart are rare and are usually secondary. Bodenheimer collected all the cases up to 1877. Leyden and Fraenkel added the subsequent cases. The possibility must not be overlooked that the so-called primary cases may arise from some undiscovered centre.

Of the 30 cases collected by Berthenson neoplasms had developed in the heart cavities 22 times. Of these neoplasms there were, of sarcoma 9, myoma 7, fibroma 6, gummata 2, carcinoma and cystic tumor 3. They were developed in the right auricle 7 times, in the right ventricle 3 times, in the left auricle 7 times, in the left ventricle 5 times. Of the 20 cases in which the sex was mentioned, 11 were men, 9 were women.

Carcinoma.—Up to the present time primary cancer of the heart has been seen 7 times. Secondary cancer is the most frequent of all the neoplasms of the heart. Thus Köhler found cancer 6 times in 9,118 autopsies, and Fanchon also found 6 cases in 8,289 autopsies. But the infrequency of this deposit may be learned from the report of Willigk, who found 477 cases of carcinoma in 4,547 autopsies, with secondary deposits in the heart 9 times and in the pericardium 7 times (Schroetter). There seems to be no particular preference as to the site of deposit. Cancer is found in both auricles and ventricles, sometimes as a single deposit, in other cases as multiple deposits scattered throughout the substance of the heart. Most of the cases

recorded, whether of primary or of secondary cancer, are soft cancers, a fact which favors metastatic deposit.

In many cases the carcinoma is an extension by contiguity or continuity of structure, as from the lungs, œsophagus, or lymph glands of the mediastinum. In rare cases these are metastases from distant seats.

Hektoen reports a secondary deposit on the wall of the right ventricle in the case of a woman, aged fifty, with metastatic medullary carcinoma of the right breast. The coronary vessels evidently furnished the avenue.

Bucquoy reported cancer of the ovaries and of the heart in a young woman, aged twenty-four.

Sarcoma.—Sarcoma of the heart is still more rare, but most of the cases of carcinoma reported to have been found in childhood were probably cases of sarcoma. The symptoms do not differ from those of carcinoma, except as regards the fact of occurrence in early life and the greater rapidity of growth. In one case Fraenkel withdrew a serosanguineous fluid from the pericardium, but there was no exudation in any one of the sixteen cases collected from the literature. This case had been recorded as a pericarditis. Sarcoma occurs, however, at any age. The sarcomatous tumors have been most commonly found in the left side of the heart, but a secondary chondrosarcoma in the anterior tip of the tricuspid valve has been reported by Ronnenschein. This tumor was the size of a walnut and, with other deposits in the body, was secondary to a chondrosarcoma of the thigh which had been exsected the year before. Jacobi reported a fibro-sarcoma of the heart, S. W. Gross a recurrent round-celled sarcoma of the heart, Hektoen a secondary and spindle-celled sarcoma developed from the wall of the right ventricle into the cavity of the heart in the case of a boy, aged twelve, affected with osteosarcoma of the tibia. The symptoms on the part of the heart were concealed by the presence of neoplasms in the neighboring regions of the lung. Notwithstanding the contraction of the right auriculo-ventricular orifice, there was no hypertrophy of the right auricle. As there were no metastases in the domain of the systemic circulation, the author believed that the germs of the neoplasm had reached the heart through the vena azygos and the superior vena cava. The same observer saw a primary epicardial round-celled sarcoma in a woman aged fifty. The tumor was seated on the anterior surface of the heart and had largely encroached upon the cavities.

A case of primary angiosarcoma of the pericardium and heart was reported by Redtenbacher. The symptoms were those of pericarditis and bilateral pleuritis with effusion.

At the post-mortem several large tumors were found in the pericardium, in the right auricle, in the right conus arteriosus, and in the anterior wall of the right ventricle. These tumors showed themselves under the microscope to be angiosarcomata. One of them had eroded the wall of the right auricle, had broken through it, and the blood from the heart had penetrated to the mass, in the interior of which was found a great bloody cavity. Through this mass enormous metastases had passed to the lungs.

Myxoma.—Myxomata of the heart have been found in nine cases, always in the left auricle, where they form nodular tumors of transparent gelatinous consistence, attached by a thin pedicle to the wall. Ribbert found gelatinous myxomata of the size of peas upon the tricuspid valves. This neoplasm is derived from the endocardium and its construction is that of branching stellate cells embedded in a gelatinous fibrous substance. In all the cases reported, the patients suffered apoplectiform attacks, which were attributed by Marchand and Robin to embolism from detached fragments in the arteries of the brain. But the malignancy is slight, for independent of emboli there are no real metastases. Salivoli reported a telangiectatic myxoma of the auricular endocardium. Berthenson describes a primary myxoma which was found in the posterior wall of the left auricle in a woman aged fifty-five, and which had produced extreme contraction of the auriculo-ventricular orifice. There had been marked protrusion of the chest and dulness over the manubrium sterni, with compression of the upper lobe of the left lung, dysphagia, and repeated lodgment of emboli in the brain, kidneys, and lungs. A diagnosis had been made of aneurysm of the arch of the aorta.

Fibroma.—Cases of fibroma were reported by Luschka, Kottmeier, and Albers. Quinquaud reported a fibrinous cyst of the heart with infarction of the spleen and kidneys; Ogle fibrinous masses deposited in the substance of the heart wall, marked in life by remarkable slowness of the pulse and paralytic seizure. Vulpian saw a fibrinous cyst with puriform contents in the left auricle; rupture took place with an attack of apoplexy terminating, with typhoid symptoms, in death.

Virchow reported a case of congenital cavernous myxoma of the heart; von Recklinghausen found in the heart of a new-born child numerous myxomata protruding partly outward and partly inward into the cavities. Hun saw lymphadenoma of the heart, Przewoski a cavernous angioma of the endocardium, Albers the only lipoma hitherto found in the substance of the heart.

Tuberculosis affects by preference the membranes of the heart, but is sometimes found as miliary tuberculosis in the form of granules disseminated through the musculature. Wagner and Waldier encoun-

tered larger caseous masses in connection with chronic tuberculosis of the lungs.

Syphilitic growths have already been described.

Parasites of the Heart.

The most common parasites are the cysticercus and the echinococcus. Mosler (1883) reported sixteen cases of cysticerci of the heart. Sometimes but a single parasite is found in the body, and that in the heart. In other cases the parasites are scattered all over the body and are deposited in various parts of the heart. Cysticerci are found most frequently in the left ventricle. They vary in size from a bean to a cherry. Frank found cysticercus in the heart and brain, Gibbes cysticercus in the heart as well as in other muscles.

Echinococci are also occasionally encountered, but of one hundred and sixty cases found in organs other than the liver the echinococcus was seen only ten times in the heart. The preference of site is rather for the right heart. The size varies from a pin's head to an orange, though Coote described a cyst in the left ventricle which produced enormous enlargement of the heart and displaced both lungs backward. The growth is usually intramural, but is sometimes pedunculated and may be in this way suspended in the cavity of the heart or detached and floated off in the circulation. Otto saw, in the right side of the heart, an accumulation of hydatids which were fixed to the Eustachian valve of the auricle by five delicate fibrinous threads. The cluster was suspended in this way a distance of one and one-half inches, extending from the auricle through the auriculo-ventricular opening into the right ventricle. The tricuspid valves were rendered incompetent. The hydatids numbered seventy or eighty, and varied in size from a millet seed to a pea. Accumulations as large as a hen's egg were reported by Meckel, Rokitansky, and Lowenhardt, and of the size of an orange by Budd and Peacock. The sac burst in six of the cases reported by Oesterlen. Budd reported hydatid tumor in the apex of the right ventricle with free hydatids in the branches of the pulmonary artery, Buck a case of hydatid cyst in the wall of the left ventricle of the heart with rupture of the cyst and sudden death, Coote an hydatid cyst in the anterior wall of the left ventricle, Bazy hydatid cyst in the interventricular septum, Crowther the bursting of an hydatid cyst of the septum ventriculi into the right ventricle; a portion of the cyst obstructed the right pulmonary artery; death occurred suddenly from cardiac syncope. Crowther saw death from bursting of an hydatid cyst of the septum into the right ventricle with occlusion of the right pulmonary artery. Vines

found a heart filled with hydatids floating in its cavity. Wilks encountered loose hydatids in the heart. Moxon saw hydatids obliterating the coronary sinus. Williams reported hydatids in the heart of a child.

Numerous deposits of actinomyces were found upon one occasion by Paltauf in the wall of the right ventricle in a phthisical subject who was supposed to be affected with tuberculous pericarditis.

Symptoms.—The symptoms do not differ from other affections which consume the heart muscle or by their presence interfere with the play of the valves. As tumors may be themselves soft or may subsequently undergo softening, and as parasites are readily detached, the accidents of embolism, including the possibility of sudden death, are not infrequent. In fact, numerous instances of sudden death in the midst of apparent perfect health have been reported in these cases. From a study of the thirty cases already reported in literature, including the thirteen recorded by Fraenkel in 1889, Berthenson concludes that the presence of atypical symptoms, especially the frequent occurrence of embolism, should excite the suspicion of a tumor growing in the cavities of the heart.

Suspicions of a diagnosis may be entertained in the presence of depots elsewhere in the body. A tumor may reveal itself by pressure signs. In a case reported by Townsend, fatal asphyxia was caused by pressure upon the pulmonary veins by a tuberculous tumor in the wall of the left auricle. Aspiration may succeed in withdrawing parasites from the pericardium or from tumors in the anterior mediastinum. In rare cases the diagnosis may be established by the character of the emboli. Oesterlen recorded the case of a girl who suffered from gangrene of the right leg caused by the wedging of echinococcus cysts into the femoral and profunda arteries, carried thither by the current of blood from a tumor in the posterior wall of the left auricle which bulged into its cavity. Maschka reported an echinococcus of the heart, causing death under the symptoms of poisoning.

The *treatment* is symptomatic or surgical. Goodhart claimed to have cured a case of hydatid cyst in the wall of the heart.

Neuroses of the Heart.

The heart beats of itself; that is, the power of pulsation (contraction) lies in the musculature of the heart itself. The contraction of the heart is not a force engendered in or derived from the nervous system or the blood. The action of the heart muscle is automatic.

The pulsation of the heart begins in the auricles and is extended

by undulation directly to the ventricles. The continuation of this undulatory movement is secured by muscular fibres which pass between and connect the auricles and ventricles. The rhythmic pulsation of the heart, though it begins at the orifice of the great veins, is not a response to the irritation of the blood circulating either in the cavities or in the vessels of the heart, but is the direct effect of automatic muscular action. For the excised heart, removed from all connection with the nerve ganglia and emptied of all blood, will still beat, and beat with true rhythm. Even fragments of heart muscle separated or distant from ganglia will continue to pulsate regularly for a time.

It is a well-established fact that the heart may execute rhythmic contractions before it possesses either nerves or ganglia. Fano demonstrated that the apex of the heart in the embryo of the chick pulsates much more slowly than the auricular end, exactly as in the adult animal, at a period when it is still without nerves.

All modern investigation points, therefore, to the supreme importance of the muscular structure itself. The heart muscle is alone the automatic motor of the circulation. It can alone accommodate itself to the various demands made upon it. It can overcome obstacles, relieve itself of distention, or compensate valvular defects, by its own inherent power and without assistance from or excitement by any influence on the part of the nervous system or the blood (His, Romberg). The heart beats because its muscular fibre is incompletely differentiated and still retains the power of spontaneous movement possessed by all primordial protoplasm (Foster). This intrinsic motion is not confined to the heart. Independent rhythmical contraction of large veins emptying into auricles has also been observed in frogs and rabbits after excision of the heart.

But mere beating of the heart does not suffice to supply the wants of the body, because the wants of the body change all the time, and the beats must change to supply the various and varying demands for blood. This change in the beating of the heart is effected by the nervous system. No subsequent disclosure has ever invalidated the declaration of Kölliker, that all automatic and rhythmical apparatus, the muscles of respiration, the heart (including the lymph hearts) in adult animals, act always under the influence of the nervous system.

Every organ is influenced in its action by two sets of nerves, the accelerator and the inhibitory. Proper function is secured only by harmonious action. Disturbance of this function constitutes disease, and if the disease is confined to the action of nerves and shows no organic changes, the disease is called a neurosis. It is impossible to understand the neuroses without some knowledge of the action of the nerves of the heart.

The nerve supply of the heart is derived from (1) cardiac branches of the vagus, which receive filaments from the spinal accessory (Ranke); (2) branches from the superior and inferior laryngeal nerves; (3) the last, often the three, cervical ganglia, and the first dorsal ganglion; (4) branches of the pulmonary plexus, chiefly on the right side, and (5) occasionally a branch from the descending part of the hypoglossus (Luschka). The heart has also ganglia of its own. These nerves from both cerebro-spinal and sympathetic systems may be regarded in action as motor, vaso-motor, and sensory, though each nerve has varied function.

According to His and Romberg, the modern anatomical idea of the innervation of the human heart is as follows: The vagus and sympathetic branches form, each after entrance into the thorax, a plexus before and behind the ascending aorta. Both these plexuses unite to form the coronary plexus, which surrounds the heart as it accompanies the coronary arteries.

The vagus controls or inhibits the heart. The vagus is also a trophic nerve of the heart (Gaskell, Eichhorst) and a few accelerator fibres probably exist in it. The vagus is often said to be the nerve which tones the heart. If the vagus trunk be irritated by an interrupted current the heart beats are rendered slow. If it be stimulated or compressed on one side (Weber, Czermak), the rhythm of the heart is altered, the contraction becomes more forcible, and the period of rest is prolonged. If the stimulant be increased, or applied to both vagi, the heart will stop in full diastole. This action is not instantaneous, but occurs after a certain period (Donders), but the action of the ventricle will reassert itself later by virtue of the power of idioventricular contraction.

The restraining or inhibitory nerves contained in the vagus trunk are really derived from the spinal accessory, while the other nerve fibres in the vagus are probably sensory. When the roots of the spinal accessory are torn out, and the nerve fibres undergo degeneration, irritation of the vagus no longer retards or stops the action of the heart.

The actual centre of the main inhibitory vagus fibres is located in the medulla, as a mass in the gray substance at the level of the point of the calamus scriptorius, but the spinal accessory arises by several roots, beginning as low down as the sixth cervical vertebra. The nerve now runs up the spinal canal through the foramen magnum into the cranial cavity, and then out through the foramen lacerum posterius in close proximity to the vagus. The internal portion of the spinal accessory subsequently joins the vagus and is distributed to the heart, presumably as its motor nerve. The vagus and the

spinal accessory then resemble a spinal nerve; the vagus with its ganglion representing the posterior or sensitive root, while the spinal accessory is the anterior or motor root (Balfour). The inhibitory centre of the activity of the heart is capable of both automatic and reflex excitement.

Disease and injury of the spinal cord and its vicinity exercises a direct influence upon the action of the heart, in that every direct irritation of the centre of the vagus must have as a consequence a retardation of the pulse. Such a retardation must also occur indirectly with every increase of the brain pressure. In the later course of the disease an increased frequency with weakness and diminished filling of the arteries signifies paralysis of the vagus centre, always a bad sign. Such an irregular and intermittent pulse is seen in the basilar meningitis of children, at a very early stage of the disease. In leptomeningitis and cervical pachymeningitis extreme retardation or increase of frequency in the action of the heart are common signs, and in progressive bulbar paralysis and amyotrophic lateral sclerosis there is often an abnormal frequency of the pulse shortly before death—up to 130, 150, and more—from paralysis of the vagus centres. The repeated attacks of syncope into which these patients fall are likewise to be referred to these centres for the innervation of the heart (Erb).

The accelerator nerves of the heart lie mostly in the sympathetic. If the nerves extending to the heart from the lower cervical and first dorsal sympathetic ganglia be stimulated, the rapidity of the heart beats is increased (v. Bezold). The centre of these nerves has not yet been definitely localized. It lies probably also in the medulla, though acceleration can no doubt be produced by centres lying high in the cerebrum (Brunton). The accelerating fibres pass downward in the cervical cord, and out by the communicating branches to join the cardiac plexus.

The frequency of the heart's action is often permanently increased in locomotor ataxia. In disease of the cervical cord the heart may be slowed to forty, thirty, or twenty beats per minute, usually only for a time. This effect may be due to a loss of the influence of the nerves, which naturally quicken the heart. In acute disease of the upper dorsal region, the heart's action may be persistently frequent (Gowers).

The innervation of the heart is effected besides by centres (ganglia) embedded in the heart itself. Recent studies prove that the ganglia belong exclusively to the auricles, that is, to the receiving chambers, and that they are connected exclusively with the sensitive sphere. These ganglia are found in the wall of the auricles, espe-

cially in the partition wall, and at the border of the great arteries. The ventricles have no ganglia. Excised fragments of heart muscle which include no ganglia will pulsate, as stated, independently, but fragments which have ganglia attached are more readily excited to contraction (Landois), probably because of the intensification or greater rapidity of perception.

In determining the work of the heart a further factor to be considered is the obstacle offered to the propulsion of the blood in the blood-vessels themselves. In this regard must be studied the resistance to the blood pressure and the resilience of the blood-vessel walls. Both these factors vary constantly, and the action of the heart must vary with them. The processes of oxygenation of blood in the lungs and of the metabolism in the tissues also exercise an influence upon the heart.

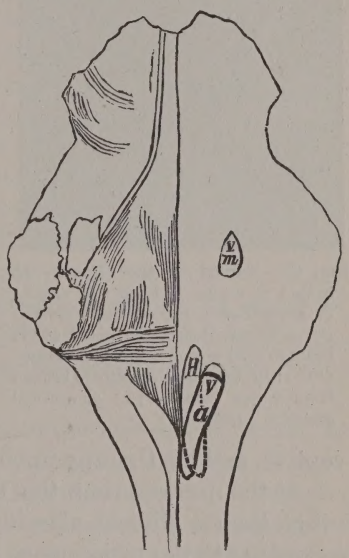

FIG. 14.—Nerve Centres in the Medulla. *v–m*, vaso-motor nucleus; *H*, hypoglossus; *v–a*, accessory vagus nucleus; the upper part forms the origin of the pneumogastric, the lower furnishes the upper fibres of the spinal accessory.

According to Starr a general vaso-motor centre lies bilaterally in the upper half of the medulla oblongata. Thus the centres for the heart, for the blood-vessels, and for respiration lie in the medulla closely related to one another, so that the pulse, the blood pressure and the breathing may all work in harmony.

The vagus and accelerating nerves belong to the efferent class, and convey impulses downward from the centre in the medulla to the heart. Thus the vagus alone may reduce the output of the ventricles, even when it is weak, as much as thirty per cent. (Balfour). But the vagus contains also sensory (afferent) fibres, which convey impressions from the heart to the medulla and produce by reflex actionsuch alteration in the contraction of the arteries and in the respiratory movement as will suit them to the needs of the heart (Brunton). Thus afferent impulses travel from the heart to the medulla, along the cardiac branch of the superior laryngeal nerve (nervus depressorius). Irritation of the central end of this nerve diminishes arterial pressure, but has no effect on the frequency or character of the heart beats (Bernstein, Burdon-Sanderson).

These points are made more plain by reference to the adjoining plate.

It is in these directions that attempts are made to understand the disturbances which distinguish the neuroses of the heart. But the difficulties to be overcome are as yet unsurmounted. The results of animal experimentation are contradictory, and studies in man are uncertain. Even under physiological conditions the relations and interdependence of the circulation and innervation of the heart and vessels are so complicated by the interactions between inhibitory fibres of the vagus and accelerator fibres of the sympathetic, between processes in the cerebral cortex, peripheral motor and sensitive nerves, and automatic centres or actions, as to defy anything like a complete or satisfactory interpretation. So that at the present time the practitioner must still rest his pathological studies largely upon symptomatology, which is apt to be deceptive, and his therapy upon experience, which is apt to be fallacious.

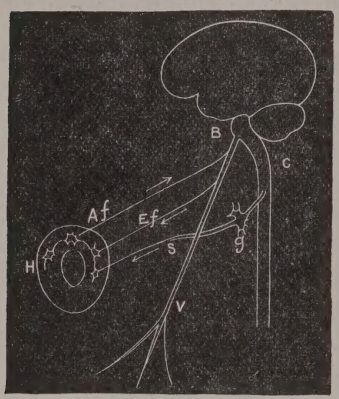

FIG. 15.—Nerves of the Heart. *B*, brain; *C*, cord; *H*, heart; *V*, vagus; *S*, sympathetic (accelerator) nerve; *Af*, afferent (depressor) nerve (from heart to vaso-motor centre in the medulla); *Ef*, efferent (inhibitory) branch from vagus (accessory); *g*, cervical ganglion. (Balfour.)

At the present time the neuroses of the heart do not admit any other than a clinical classification, and the diseases are discussed according to the prominence of individual symptoms, weakness, palpitation, arhythmia, pain, etc.

NEURASTHENIA CORDIS.

Neurasthenia cordis, neurasthenia vaso-motoria, weak heart, irritable heart (Da Costa), or cardiac insufficiency, is a term applied to the heart to explain heart failures or insufficiencies by defects of innervation. The true neurasthenia must therefore exclude all organic disease of the heart, at least of the heart muscle. In the light of present knowledge it is questionable if such a condition exists or if affection of nerve force can be isolated in its action. The assumption that a weakness which is transitory must be nervous and functional, while that which is permanent implies impairment of the structure of the heart, will not bear analysis, as many cases of weak and irregular action depend upon the influence upon the heart muscle of toxins (*e.g.*, uric acid) which are subsequently eliminated from

the body. Insufficiency from muscular cause belongs to the chapter on diseases of the heart itself.

A neurosis is always said to be a purely functional disturbance, and the existence of a lesion is said to exclude the pure neurosis. This view may be no longer accepted in our day. A change of function implies always a change of structure. A failure to discover the alteration is simply a reflection upon the means or acuteness of observation. Hence modern pathologists and clinicians prefer to say a neurosis is a condition "without demonstrable lesion." But even this statement must be modified, upon the recognition of changes under more searching investigation, especially as we acquire more thorough knowledge of the construction of the normal nerve cell. Thus degeneration, granulation, shrivelling of protoplasm, and especially vacuolation of nuclei have been observed and described in the nerve cells after fatigue and after direct and reflex irritation, by Hodge, Sadovski, Ternowski, Whitwell, and Korybutt (Rachford). These changes were none the less structural because transitory, as the changes which are transitory at first become permanent later. Most interesting and satisfactory was the condition found by Arndt of arrest of development of the ganglion cells at the fœtal stage, originary agenesis of nerve fibres, defective development of the nerve sheath (Krafft-Ebing).

Interesting in this connection are the investigations of Marie de Manacéine, who kept ten young dogs awake for a period of two, three, and four months. Four of the dogs died when all sleep was prevented in the course of ninety-two to one hundred and forty-three hours. The temperature sank during the second twenty-four hours 0.5–0.9° C., and continued to sink up to death, when it was 4° or 5° below normal. The reflexes became feebler, the reaction of the pupil more sluggish; the number of red blood corpuscles sank quickly from five to two millions, but they increased in the last twenty-four hours, probably in consequence of desiccation of the body. In contrast with the condition found in starvation, the dogs killed by insomnia showed more profound alterations in the brain than in any other organ. Numerous ganglion cells were found in a state of fatty degeneration; the blood-vessels were often surrounded by a thick layer of white corpuscles. Small capillary hemorrhages occurred on the surface of the cortex, larger about the optic nerves and in the substance of the thalami optici. The weight of the whole body was reduced only from five to thirteen per cent. After the insomnia had lasted from ninety-six to one hundred and twenty hours, it was found impossible to rescue the animals either by warmth, artificial nutrition, or abundant sleep.

Pöhl believed that the products of metabolism were eliminated by oxidation and that six-sevenths of the oxygen was furnished by the atmospheric air and one-seventh by means of a ferment produced in the body. This ferment is spermin, which is found everywhere in the blood, especially in the testicle, prostate, thymus and thyroid glands, and is probably formed in these organs. When this spermin is not formed or is limited, auto-intoxication occurs. Thus the basis of neurasthenia lies upon a disturbed chemical action of the nerve cells, and in this way is explained the frequency of uric-acid diathesis, phosphaturia, oxaluria, etc., in neurasthenic states.

The diagnosis of neurasthenia cordis may be accepted only in cases of general neurasthenia which show elsewhere evidence of exhaustion of the nervous system. Cases, too, of reflex irritation and exhaustion, whether acute and profound as from shock, surgical or psychical, or more protracted or insidious as from disease of a distant organ, or chronic mental depression, may also fall under this head. Cases of sclerosis or of ascending neuritis which impair the action, or absolutely arrest the movement of the heart, by final implication of the cardiac centres in the cord, do not fall in the domain of pure neurasthenia.

Patients affected with neurasthenia cordis are usually young, under middle age, and are generally of good constitution, but belong to the neuropathic class. The neuropathy is, for the most part, an inheritance. It rests upon defective endowment. But it is not infrequently acquired; in men, especially by overstrain at study, failures in business, bad habits, sexual excesses, including onanism, and as Seeligmüller emphasizes, habitual sexual excitement without satisfaction, above all by the many forms of coitus reservatus; in women, by disappointment in love affairs, social agonies, domestic infelicities, etc. Lehr says that students at the last end of the session, who consume the night in study, sustain themselves with tobacco, strong tea, and coffee, form twenty per cent. of cases of neurasthenia, while forty per cent. is composed of merchants and artisans who have used themselves up in unsuccessful rivalries and struggles. The relative frequency of neurasthenia cordis in general neurasthenia and the form of the affection is well shown in the following table furnished by Lehr:

	Males.	Females.
Whole number of cases	118	47
Increased action of the heart	71	32
Increased reflex irritability	78	33
Neither increase	23	8
Reflexes normal	17	6
Pulse normal	20	5
Pulse retarded	4	2
Increased action as the chief symptom	35	12

Pronounced forms are also left after the acute diseases, such as typhoid fever. The uric-acid diathesis, so-called, is a pretty regular concomitant of neurasthenia, and good authority could be cited for regarding neurasthenia as a form of suppressed gout.

More than half the cases of general neurasthenia show evidence of nervous heart weakness. There are cases of pure neurasthenia in which the nervous symptoms predominate, others in which the heart symptoms are most distinct. Rosenbach claims that men are more frequently affected than women, and Lehr's tables seem to confirm this view; but in the limited experience of the writer the reverse is true to such degree as to lead to the belief that in many cases neurasthenia in women has been regarded simply as an hysteria. Though full brother to hysteria, neurasthenia is, however, by no means a loss of control of the will. On the contrary, many patients are strong-willed and, as already remarked, the condition is held by competent authorities to be more common in men. In fact, the tendency of the disease is rather toward hypochondriasis than hysteria.

Symptoms.—The disease begins usually gradually, sometimes suddenly, with a stage of excitation. There is frequent change from pallor to flushing of the face, attacks of sudden coldness of the hands and feet, which shortly afterward become burning hot. There is often a sensation of numbness in the fingers and toes from ischæmia. Patients say parts of the body feel as if they were dead. Sometimes there is a marked sensitiveness in the region of the heart and intercostal spaces, as in intercostal neuralgia. The heart itself shows nothing abnormal save that in certain cases its action is stronger, and in others more rapid. The attack often begins with increased frequency of the pulse. This increased action shows itself first after excitement, or after the use of stimulants, such as coffee, tea, alcohol, etc., and may amount to a genuine tachycardia, with a frequency of 180 to 200 beats in the minute. Retardation is much more rare. The increased rapidity is seen especially after the slightest effort or psychical excitement. Sometimes the mere thought of an effort excites the pulse. The patient becomes exceedingly sensitive; a noise, the entrance of a stranger, the simple raising of the body to the sitting posture excites the pulse. With the increase of frequency there is also irregularity, intermittence. Real attacks of palpitation are rare, but the patient complains of the feeling even though the action of the heart is less than normal or the impulse is imperceptible. The heart sounds are usually feebler. Under normal condition of the heart strong pulsations are felt in individual regions. There may be dilatation of the arteries and abnormal tortuousness, especially of temporal arteries. There is a feeling of fulness of blood in the head;

the surface is reddened in spots or streaks. Further, there may be severe precordial anxiety with an indescribable oppression simulating angina. Sleep is restless, or the patient is unable to fall asleep or awakens with a sudden jump of the heart or other expression of anxiety.

This stage of irritation now passes gradually into a state of depression, wherein the nutrition suffers, the color becomes pale, or the face is constantly flushed, is sometimes blue, especially on exposure to cold; insomnia is constant. The disposition is changed, and the patient becomes irritable or more frequently apathetic. In many cases the patient seems to be in a constant state of suppressed excitement.

During the irritative stage the heart muscle is still intact; the paretic stage is expressed by irritable weakness with corresponding weakness and excitability of the pulse. The atonic stage is further distinguished by dicrotism of the pulse. Dicrotism depends upon diminished tension of the arteries, and as this condition is universal, it must be referred to the centre of vaso-motor action. The atonic form of nervous heart weakness depends, therefore, upon temporary paresis of the vaso-motor centre in the medulla (Lehr).

The following are typical tracings of the irritable and exhausted stages, showing the deviations from the normal curve:

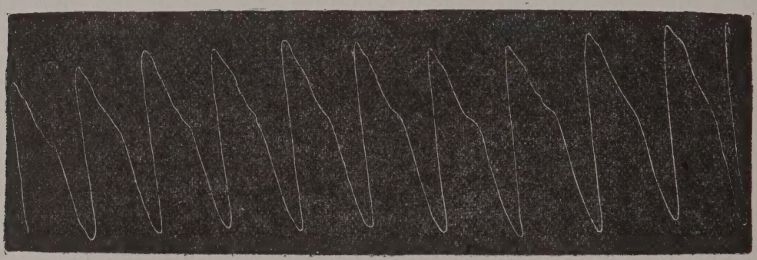

FIG. 16.—Neurasthenia; irritable stage, showing strong action of the heart with resistless arteries.

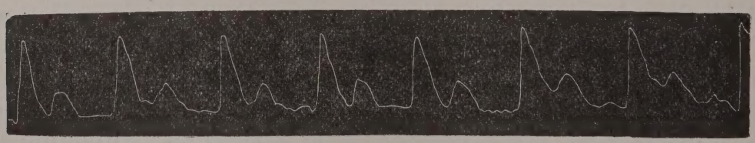

FIG. 17.—Neurasthenia; atonic (transition) stage, showing dicrotism.

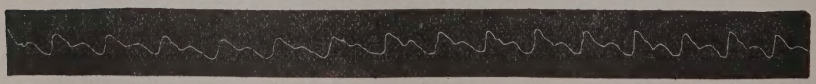

FIG. 18.—Neurasthenia; paretic stage, showing feeble action of the heart and undistended arteries.

Diagnosis.—Neurasthenia is a dangerous term, as it easily serves as a cloak to cover organic affections. The diagnosis, which should be reached by exclusion, rests upon the characteristic depression in the psychical sphere, the lack of interest in the surroundings, the irritability of disposition, the agoraphobia, monophobia, nosophobia, the suppressed excitement, the constant introspection, the general weakness of the whole body, disinclination to effort, the increase of the reflexes, and the long duration of the disease without evidence of organic lesion (diabetes, carcinoma, tuberculosis, Addison's disease, brain syphilis, etc.). The neurasthenic patient feels himself the change in his character and capacity, which he regrets and laments and constantly compares with his former state. In this regard the condition is distinguished from organic affections of the brain, as in dementia paralytica, where the patient is not cognizant of the change in his character and disposition.

The diagnosis necessitates further separation from hysteria, hypochondriasis, and, on the part of the heart, from failure due to organic disease.

The differentiation of hysteria is not a matter of the recognition of a disease, but of distinguishing an individual symptom, whether this or that symptom is of hysterical origin or nature. The most essential thing in excluding hysteria is the recognition of the symptoms of organic disease. Most organic diseases have symptoms which hysteria may imitate, but they have also symptoms which hysteria cannot imitate. Thus hysteria may imitate emaciation, cough, pain in the chest, and even the expectoration of blood in tuberculosis. But hysteria cannot present the fever, the expectoration, the bacilli in the expectoration, and the physical signs of the disease (Wagner). Then, while hysteria may imitate diseases at the start, it may be easily eliminated in the further course of a malady. Thus hysteria may present at the start the symptoms of an insidious heart disease, myocarditis, valve lesion, etc., but the conditions are easily separated later. Certain symptoms belong more definitely to hysteria, *e.g.*, the paroxysmal attacks, the emotional dramas, sensory hemianæsthesia, certain hyperæsthesias, clavus, globus hystericus, hysterogenic zones, etc.

Hypochondriasis, which is closer to neurasthenia, is separated by the fact that there is, or need be, in it no weakness at all, no asthenia. Many, indeed most, hypochondriacs are strong in body and mind. In neurasthenia the feeling of weakness is real; both the patient and the physician appreciate it as real; in hysteria and hypochondriasis it has no foundation in fact and both the patient and physician know it to be feigned, exaggerated, and false.

In so far as the heart is concerned, the separation of neurasthenia from organic disease is established by the absence in neurasthenia of any increase in size, or of murmur save that which may be attributed to anæmia. The anomalies in frequence and rhythm vary from time to time in the course of neurasthenia; they occur often only in attacks or paroxysms, which may be attributed to emotional excitement, intellectual effort, errors in diet. Bodily effort which intensifies the symptoms of organic disease has little or no effect upon neurasthenia. The main point of distinction lies in the fact that neurasthenia leaves no permanent impression, as patients recover with a full *restitutio ad integrum*. Further, the symptoms on the part of the heart are associated with the evidence of excitation or exhaustion of the brain, stomach, etc., cerebrasthenia and nervous dyspepsia. Irritable weakness is the stamp of neurasthenia.

The *prognosis*, in so far as life is concerned, is good, and is under all circumstances better than in asthenia of organic (muscular) origin. In a general way, it may be said that even the severe and protracted forms seldom lead to permanent injury. The attacks cease sooner or later, sometimes more or less suddenly, and generally with the improvement of nutrition and the securing of sleep. But the prognosis in chronic cases, so far as complete restoration is concerned, is not so good. Here the outlook will depend largely upon the ability of the practitioner or patient to remove or relieve the cause. Bad habits may be broken up; a man may be given a new start in life, and a woman may be reconciled by religion, philosophy, or finally by consuetude, to irremediable evils. For failures in business there is sometimes stimulus in a new start, sometimes content in resignation to fate. For disappointment in love affairs the real relief is substitution, as "one nail drives out another."

The *treatment*, which is that of neurasthenia in general, is more a matter of management than medicines, and will address itself with particulars to the individual case. The best cure is the rest cure with isolation, and of necessity in a proper institution away from home; but what avails the rest cure under monotonous or melancholic surroundings? If the rest cure is the best cure, the best tonic is pleasure, and here it is especially that what is food to one may be poison to another. To many women and to most men idleness is not pleasure and stagnation is not rest. In these cases the rest cure may be a torture. The nervous system is best fortified by self-denial, and as soon as possible by occupation of mind. The patient who is inclined to become a recluse may be led out of this state by cheerful society, or may have the mind diverted by the panorama of travel. Music, especially the making of it by the

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patient, often gives relief. But the main thing is the drill and discipline of mind and body, which comes with the routine of daily work.

Exhaustion of the heart nerves, as of other nerves, is best relieved by food and sleep, and tone to the heart is best furnished by cold baths and by properly graded exercise. The appetite may be increased and the digestion improved by daily irrigation of the stomach with a soft tube, by the administration of tonics, the tincture of nux vomica, by arsenic, Roncegno water, or diluted hydrochloric acid. The uric acid diathesis is combated best by the free use of alkalies, Carlsbad salts, etc. A bitter or aromatic tincture of cascara or rhubarb will relieve the constipation, which is almost always present and which much increases the tendency to hypochondriasis.

Insomnia aggravates the condition and intensifies all the symptoms. The heart is especially weak in the morning after a night of unrest. In the relief of insomnia it is difficult to steer between Scylla and Charybdis, as there is great danger—though not so great as in hysteria—of forming habits with hypnotics. The quieting effects of the warm bath at bedtime should be tried first. It may be necessary at times to administer the mildest and safest of the hypnotics, trional, in dose of grs. xv.–xxx. in a cup of hot tea or milk, or chloramid, grs. xx.–xxx., with an ounce of the compound tincture of cardamom in a wineglass of water, at bedtime. Neither of these remedies is likely to produce a habit. Cocaine has high commendation with some neurologists. It is administered in dose of $\frac{1}{4}$ to $\frac{1}{3}$ of a grain (0.016–0.02) twice a day. The author has never got good from it and the danger of engendering a bad habit must be kept constantly in mind. It must be an extreme case—like that of an obstinate melancholia—which would justify the use of opium in any form.

Jules Chéron reported a number of cases in which neurasthenia was successfully treated by the subcutaneous transfusions of artificial serum at the hospital of St. Lazare, in Paris. Ralph Browne speaks enthusiastically of this method of treatment as a means of increasing the vitality of the entire individual and "raising of power of action of all the systems of the human organism." He declares that the injection under the skin of a few cubic centimetres of serum is followed by "a sensation of well-being, of energy, of greater vitality." While it is a well-established fact that the transfusion of salt water, 0.6 per cent., relieves states of exhaustion, it is not likely that this method of treatment could reach the real cause of neurasthenia and hysteria, diseases of the nerve cells of the cortex of the brain. The statements of these observers are too vague and too full of glittering generalities for the requirements of modern science. In quite recent times nuclein or protonuclein, as it is sometimes called,

has been empirically recommended in the treatment of neurasthenia. The writer has had but little experience with this agent, and this experience has not been at all encouraging. Finally, there is the field of hypnotism, which seems to apply to individual cases, selected with great care, but in recklessly relying on it both the physician and patient are more likely to wander away from the truth and thus deeper into the obscurities of neurasthenia and hysteria.

The heart is best toned by strychnine, especially in subcutaneous injection, and the remedy is best given in the form of the nitrate of which the dose should be small, gr. $\frac{1}{100}$ at first, to be increased gradually to gr. $\frac{1}{40}$ once or twice a day. Strychnine heightens the reflex excitability of the centres of the heart nerves in the spinal cord. The cold bath with brisk friction of the skin in the morning makes powerful appeal in the same way. Lehr found hydrotherapy, in the form of the cold douche with frictions, the most effective of all means in restoring tone to the heart. The immediate effect of the bath is shown in the disappearance of dicrotism with the increased tension of the arteries.

Life in the open air, associated with exercise, a sea voyage, riding, boating, mountain climbing, feeds the nervous system with oxygen and blood, and nothing so effectively rouses lethargic centres in the brain and cord as floods of sunshine and oceans of fresh air.

Palpitation.

Palpitation is a beating of the heart which is felt by the patient as an unpleasant or distressing sensation. Mere beating does not constitute palpitation, and violent beating is not sufficient. The heart beats violently under effort. The increased action which occurs under effort, running, climbing, etc., is physiological. Palpitation is pathological when it occurs without any effort at all. "Palpitation of the heart may be defined," said Laennec, "as a beating of the heart which is felt and which disturbs the patient. Whatever the cause, all palpitations have the common character that the patient feels his heart beat." Laennec declared that he had often heard his own heart beat in lying down, and that he could frequently distinguish the alternate beating of the auricle and ventricle. He made these observations on himself especially in conditions of nervous agitation and insomnia.

Palpitation of the heart, though a very distinct symptom, is in no sense a disease; it is the symptom of many diseases, and though very similar in its manifestations, has often a very complex cause. It is not possible to define the exact mechanism of palpitation. It

may depend upon irregular action of the heart muscle itself, excitement of the accelerator nerve, or suppression of the action of the inhibitory nerve. Finally it may depend upon alteration in the quality or quantity of the blood, or in the walls of the blood-vessels. The inter-relation of these various factors is, as already stated, so complicated and sensitive as often to make impossible a true interpretation of an individual case.

Nervous palpitation, which is alone here considered, independent of demonstrable or organic disease of the heart, is an affection more particularly of the young or middle-aged and of the nervous temperament. Palpitations at the extremes of life are dependent, in youth especially—aside from the anæmias—upon rheumatism with organic disease of the heart, and in old age upon atheromatous and cirrhotic processes in the heart and blood-vessels and kidneys. Nervous palpitation is an affection also of the better classes, and is more common among, though by no means confined to, those of indoor life, sedentary habits, and luxurious living.

Most cases of palpitation can be reduced to three causes: direct, reflex, and toxic. These causes are often conjoined; they rarely operate singly or alone.

Under the direct causes are irritation at the origin of the nerves, as organic disease of the brain, including the finer lesions of the cortex. Palpitation sometimes occurs as an alternating neurosis, with migraine or with epilepsy; in fact, palpitation is sometimes the aura of epilepsy.

The action of the heart under psychical influence is, up to certain limits, physiological. Beyond these limits, or when the action of the heart is felt by the patient, it is pathological and constitutes palpitation. Thus, the heart is said to beat high, to leap, in conditions of exaltation, joy, anger, expectation; or it flutters with fear, fright, anxiety. The heart is sensitive to and responds to every emotion:

> Etwas wünschen und verlieren,
> Etwas hoffen muss das Herz;
> Etwas zu verlieren bangen (Rückert).

Nervous patients are seized with palpitation under examination. Many individuals cannot pass for life insurance until they have remained some time in the consultation room, and the heart's action has become quieted. Stage fright often takes the form of palpitation. Public speakers have not infrequently fallen victims to the tumultuous action of the heart, which breaks distant blood-vessels in the brain, or leads to over-strain and paralysis of the heart itself. The writer is consulted every year by students affected with palpitation dur-

ing the study of diseases of the heart. Peter Frank had such violent attacks, with intermittent pulse, while he was writing his work on diseases of the heart, that he believed himself to be the subject of aneurysm. After he had finished his work, and sought diversion in travel, the attacks disappeared. Here also come in the cases of palpitation which occur under the influence of depressing mental emotions of long duration, where the increase of excitability is so great that the slightest irritation will set up tumultuous action. The most important factors in the production of palpitation are psychical conditions, grief, shock, desire, apprehension, anxiety, homesickness, love sickness, embarrassment, etc., more especially the depressing emotions. The "broken heart" is often preceded by a long period of palpitation.

Palpitation also occurs in organic disease of the cord, implicating the nerves of the heart at their origin, thus as an attendant upon or precursor of lateral sclerosis in its later stages, bulbar paralysis, or exophthalmic goitre. Pressure upon the vagus in its course may also produce palpitation. Proebsting collected five cases, including three of Basedow's disease, in which the autopsy showed pressure upon the vagus by ganglionic swellings, and Chevalier appealed to the observations of Riegel and Pelizäus to prove that caseous tuberculous glands often caused acceleration of the heart with palpitation. Under the direct causes should be included also affections of the terminal filaments of the vagus and of the heart muscle itself. The palpitation which occurs under the influence of amyl nitrite is due to paralysis of the terminal filaments of the vagus in the heart, whereby the inhibitory influence is cut out. The palpitation with other disturbance which arises in consequence of affection of the heart muscle, hypertrophy, valve lesion, etc., is evidence of organic disease and is not considered here. But the purely nervous cases preponderate by far. Every general practitioner will agree with Gerhardt when he declares that more than half the people who consult him for relief of palpitation of the heart suffer, not from valve disease, but from disturbance of innervation. Lehr found that 103 of 165 patients whom he saw in a certain period suffered only from functional disturbances, and that 47 of these 103 patients suffered only from palpitation in the entire absence of any other symptom or lesion. Of the 750 cases of chronic heart disease under the observation of Schott, only 245, that is, one-third of all the cases, were affected with organic valve lesion.

The reflex causes are various diseases of other organs, as of the stomach. Dyspepsia, from whatever cause, is often marked by palpitation. The condition may be caused here by direct—*i.e.*,

mechanical—pressure of the distended stomach, and by mechanical occlusion of the great vessels. It is more frequently toxic, and results from the absorption of toxic matter through the alimentary canal. Thus palpitations are found in connection with sick headaches. Diseases of the uterus and its appendages are frequently connected with palpitation, the proof of which is the relief of the condition with the cure of the disease. One of the worst cases in the experience of the writer was cured by exsection of a diseased Fallopian tube. Intense palpitation was caused once in a case, also under the observation of the writer, as the result of irritation of the prostatic urethra. The palpitation awoke the patient at night with a sense of impending death, and in a cold sweat. Posterior urethritis has in its train of symptoms headache, neuralgia, hysteria virilis, and at times palpitation of the heart. Here, too, the condition is partly, if not chiefly, toxic, in that toxins are absorbed into the blood and directly—*i.e.*, through the coronary arteries—irritate the heart muscle. With the elimination of these toxins, mostly by diuresis, the palpitation ceases; or the attacks are prevented altogether—*i.e.*, the condition is cured—by deep injections with strong solutions of the nitrate of silver. The connection between the heart and the genital apparatus is intimate. Palpitation frequently follows sexual excess, and is an even more common attendant upon perversions and unnatural or incomplete gratifications. On the other hand, palpitation may result from excessive repressions. Fothergill reports the case of a man of active inclinations whose wife had died suddenly. In this case the repression gave rise to violent paroxysms of palpitation, which were at once relieved by intercourse. The palpitation in this case was, however, rather the suppression of a habit than the effect of continence, for there is nothing in habitual continence in any way injurious to health. Fothergill dilates upon the relations of the heart to the reproductive organs, finding in this connection a liability to palpitation at puberty, and still more at the menopause. In one case, in a young gentleman, the cause of the palpitation was long sought for in vain, until at last rectal irritation was discovered; appropriate treatment directed to the cause of the irritation entirely relieved this reflex palpitation. Finally diseases of the ovaries or of the adrenals, or attacks of colic produced by the passage of gall-stones and kidney-stones may induce attacks of palpitation.

Under toxic causes may be included impoverishment of the blood. Palpitations occur in typical outbreaks in cases of anæmia of all forms, including that from hemorrhage or other exhausting discharges. These cases are often expressions of mere exhaustion or insufficient nutrition. They are also due to toxic products which

develop in the blood in these states. So tuberculosis and Bright's disease are prefaced for a long time by attacks of palpitation, and are accompanied by them through their course. The heart, in these conditions, becomes excitable under the slightest effort, or under no apparent effort. Such individuals are said to be sufferers from erethismus cordis. The frequency of palpitation as an expression of incipient lung trouble is emphasized by Hirtz with the aphorism, "If there are palpitations, auscultate the lungs; if there is suffocation, auscultate the heart." Here, too, there is an insufficient nutrition, as well as a toxic action. Absorption of diseased products from various organs and from the alimentary canal has already been mentioned. The point is that in so many cases palpitation depends upon a poisoning of the heart muscle itself, as well as of the nerve centres.

Palpitation occurs in Bright's disease, especially in connection with hypertrophy of the heart, and repeated attacks of palpitation in middle life should excite inquiry for the other neuroses, the headache, hebetude, vertigo, etc., of this affection. Evidence of this condition is revealed, also, by the increase of blood pressure in the arteries. Attacks of palpitation are frequent in gout, due partly to the action of toxins (uric acid), partly to the condition of the blood and disease of the walls of the blood-vessels. The palpitation which occurs in rheumatism is to be attributed for the most part to organic disease. Attacks of palpitation occur in all infectious diseases.

Alcohol is one of the most frequent causes of palpitation. Parkes showed that the pulse of a man who drank only whiskey for six days numbered 90.9 in the minute, while that of a man who drank only water in the same time beat only 73.6 per minute. Coffee and tea produce the same effect, but in much less degree. Tobacco is a powerful poison to the heart. The first use is wont to produce attacks of palpitation. The habitué experiences palpitation as well as pain.

Palpitation occurs in consequence of marked increase of the quantity of blood. Where the increase is physiological, as in the process of gaining weight, or pathological, as in loss of resilience with dilatation of the blood-vessels, the heart is hypertrophied, and overcomes the obstacle without effort. Where the increase or change of pressure is temporary, as after meals, after exposure to heat in a Turkish bath, after removal of fluid in ascites or pleurisy, the regulatory apparatus is disturbed and palpitation occurs. With each contraction the ventricles probably empty themselves completely, and from each one about 180 grams of blood (Fisk, Volkmann, Vierordt) are forced into the arteries against an arterial pressure equal to 150

mm. Hg., and while variations of arterial pressure from 58 mm. to 147 mm. Hg. have practically no effect whatever on the quantity of blood sent out from the ventricle at each systole (Thompson), they may for a time disturb the action of the heart and produce palpitation. To show the effect of conjoint (toxic) cause, the palpitation after meals has been attributed to the overloading of the blood with peptones.

Symptoms.

The main symptom is obvious and obtrusive in the tumultuous action of the heart, which varies in every degree of intensity. Sometimes the patient feels and is frightened by the violence of the throbbing, fearing often that the heart will burst its bounds. The heart hammers upon the chest-wall and sometimes agitates the whole upper part of the body. Palpitation occurs, as a rule, in paroxysms, or is excited by some trivial cause, a feeling of apprehension, a noise in the street, a knock on the door, the arrival of a dispatch or a letter; mere waiting or expectation may excite an attack. Sometimes the palpitation is more directly connected with physical effort, with an attack of indigestion, overfilling of the blood-vessels, etc. Many people suffer palpitation immediately after meals. Certain individuals are attacked with palpitation whenever they lie upon one side or the other, especially upon the left side.

The palpitation is often subject to intermissions, during which the heart seems to stand still. Fothergill comments upon the complaint of patients who declare that "It is not the palpitation which frightens me so, for then I know the heart is going; it is the sudden stoppage which alarms me." Hearts of this description he distinguishes as "badly behaved" or excitable. Bouillaud spoke of such an attack as a "folie véritable." Sometimes the heart seems to jump in its action. Some patients speak of a peculiar "roll over" feeling. Others experience "choking sensations and a feeling as if the heart were jumping into the throat" (Walshe). The attacks are often attended with dyspnœa, with deep and sighing respiration, broken or interrupted speech, a feeling of anxiety and constriction, ringing in the ears, sparks before the eyes, headache and vertigo, tremor and agitation of the whole body. In severe cases there is a feeling of great anxiety and even of imminent death. The face is cool, pallid, sometimes livid, and cold sweat stands upon the forehead. There may be tremor of the extremities and a tendency to syncope. After the attack there is often an indescribable depression, with languor and weariness. During a milder attack, especially of gastric origin, there is sometimes distinct boulimia, excessive hunger, which is

relieved by the taking of food, but is followed later by eructations and other signs of dyspepsia.

In purely nervous cases the patients may show a good color and a good condition of nutrition for a long time, so that the appearance may stand in marked contrast with the complaint and distress.

The attacks occur usually in the daytime and last from a few minutes to the greater part of an hour, or off and on during the greater part of a day. Sometimes the patient is awakened suddenly from sleep with more or less violent palpitation, which causes indescribable anxiety and apprehension, the rest of the night being often sleepless from dread of a repetition of the attack. Attacks of palpitation confined to the night should excite suspicion of epilepsy.

The heart itself in purely nervous palpitation shows no sign of disease. There is no increase of dulness and no murmur. Yet frequently repeated or violent attacks may in the course of time produce some enlargement with subsequent final dilatation, and murmurs may mark a coexisting or causative anæmia or chlorosis. The valve sounds are clear in uncomplicated cases or are accentuated often to the degree of *cliquetis métalliques.*

Diagnosis.

Inasmuch as palpitation is only a symptom, the existence of it is recognized by the patient, and the practical interest of the diagnosis concerns only the discovery of the cause and the exclusion of organic disease. Nervous palpitation occurs most frequently in anæmia and neurasthenia and under the action of irritants, alcohol, tobacco, tea, or coffee, excesses, or bad habits, and attacks are precipitated often by emotional causes. Organic disease of the heart is excluded by the absence of any increase in size and by the absence of bruits, save those which pertain to anæmia, chlorosis, etc. A systolic murmur of anæmia may coexist with palpitation, but a diastolic murmur, which signifies organic disease, excludes nervous palpitation.

Prognosis.

Inasmuch as pure nervous palpitation is independent of any organic disease, the prognosis is for the most part good, but depends upon the ability of the physician to discover, or upon the ability of the patient to surrender the cause of the condition. Any anatomical lesion aggravates the prognosis. Any marked irregularity of action is more serious than mere palpitation, however violent and distinct; intermission in turn is more serious than irregularity. In all cases, however, the prognosis of nervous palpitation is more favorable than

that from organic disease of the heart, brain, blood-vessels, or kidneys. The exclusion of affection of these organs is sometimes difficult, and the prognosis should be made with caution until repeated and critical examination has made the diagnosis sure.

Treatment.

The treatment must address itself to the cause. Anæmia, chlorosis, may be relieved by iron, arsenic, food, and fresh air. Bad habits are to be overcome; the use of alcohol, tobacco, coffee, and tea must be abandoned. The treatment of neurasthenia has been prescribed. The patient must himself "free from memory the written sorrow." Disturbance of digestion may be regulated by diet, or relieved by the use of the stomach-tube, dilute hydrochloric acid, and bitter tonics. Plethora, gout, may be reduced by diet and exercise and by the use of saline laxatives, Carlsbad water. Constipation may be overcome by the vegetable laxatives, cascara, rhubarb, podophyllin.

During the paroxysm of palpitation the patient should observe the semi-recumbent posture, the chest should be bared or relieved of superfluous clothing, the windows should be open to admit fresh air, and all sources of irritation or annoyance removed as much as possible. An ice-bag or cloths wrung out of ice water applied to the region of the heart may quickly still the tumultuous action. The administration of stimulants may have the desired effect. Thus half a teaspoonful to half a tablespoonful of good French brandy, or aromatic spirits of ammonia, gtt. xx.–xxx., or half a teaspoonful of Hoffmann's anodyne, may control or cut short the attack. Excessive nervousness may be relieved by sodium bromide, grs. xx.–xl. in half a glass of water, or in a more extreme case by chloral grs. v.–x. or cocaine gr. $\frac{1}{4}$–$\frac{1}{2}$, or finally by morphine, gr. $\frac{1}{4}$. The constant current of electricity has sometimes proved of value. The positive pole should be applied to the vagus at the inner surface of the sterno-mastoid, the negative pole to the lower border of the sternum. Currents of moderate strength are used under daily sessions of five minutes' duration. Counter-irritation to the surface, as by a sinapism, will generally suffice to relieve any associate pain. Much comfort may be got sometimes from the application of a good belladonna or other plaster that will stick tight.

After discovery and removal of the cause, as by discharge of gall stone, curetting of the uterus, cure of gonorrhœa, gastric catarrh, fixation of a floating kidney, etc., any remaining lack of tone in the nerves of the heart may be fortified by hydrotherapy. The daily

morning bath, tepid at first and gradually cooler, with subsequent friction of the chest, stimulates the heart through the nerves of sensation and respiration. Graded exercise will build up the heart muscle. Strychnine, subcutaneously, will heighten the inhibitory tone. The excitement of exhaustion may be quieted by the tincture of digitalis in dose of gtt. x. every four hours; or, if this drug be contra-indicated from any cause, by the tincture of strophanthus in the same dose. Quinine is always a good heart tonic.

Tachycardia.

Tachycardia (*ταχύς*, quick, *καρδία*, heart), more properly polycardia or pyknocardia (*πυκνός σφυγμός* of Hippocrates), heart hurry, is a comparatively new term introduced by Proebsting, a pupil of Gerhardt (1881). The condition was included in old times under palpitation. The distinction is now drawn between the two in that palpitation is a beating of the heart that is felt by the patient, while tachycardia is an increase in the frequency of the beats. Tachycardia like palpitation is always only a symptom and never a distinct disease. In palpitation there is usually, but is not necessarily, an increase of frequency; but the heart may throb violently, and yet may beat slowly. Tachycardia is usually, but is not necessarily, perceived by the patient. Sometimes the condition is recognized by the physician or by the patient only when he counts the pulse at the wrist.

Sometimes the heart beats naturally fast. Thus the pulse of Sir William Congreve was said to have been 120 per minute. The term or condition has come to be limited in our day to that rapid beating of the heart which occurs in paroxysms.

A temporary or a permanent or habitual tachycardia may result from many causes. Thus the pulsations of the heart are increased in frequency as the result of exercise, and the pulse usually keeps pace with the temperature in all cases of fever. In exhaustion from any cause, in anæmia and chlorosis, after hemorrhage, after postpartum hemorrhage, in the course of chronic disease, tuberculosis, in the convalescence from acute diseases, in weakness of the heart muscle itself, that is, in all cases of insufficiency, the action of the heart is increased in frequency to make up for defects in force in the attempt to supply the demands of the tissues for blood.

But tachycardia as a neurosis must be independent of any cause of this kind. True tachycardia results from a disturbance in the regulatory apparatus and implies a defect of innervation. This defect may be an overstimulation of the accelerator (sympathetic) nerves, or a paralysis of the inhibitory (vagus) nerves.

As there is no knowledge further than that furnished by experiment as to the action of the accelerator nerves, it is assumed that the essence of tachyardia probably lies in arrest of action of the vagus nerves, which lets the heart run away with itself "like the works of a clock when the pendulum is taken off."

It is thought that, taking everything together, most of the nervous tachycardias may be referred to paralysis of the vagus; those with high pulse frequency, over 200, to a combination of paralysis of the vagus with irritation of the sympathetic; only a few of the cases, especially those of lighter form, to irritation of the sympathetic alone. Irritation of the accelerator nerve in animals, not poisoned in any way, increases the pulsation of the heart from thirty to seventy per cent., but not more.

Double section of the vagus does not greatly increase the pulse frequency. According to v. Bezold, it may be increased to 120–180 beats. The investigations of Martius showed that when the influence of the vagus was entirely abstracted from the human heart, the increase in the pulse frequency did not exceed moderate limits, that is, the pulse was not increased more than to 150 to 160 beats. The subject may be put then as follows: Increase to 120 beats—that is, thirty to seventy per cent. of the normal number—implies irritation of the sympathetic; increase to from 120 to 180 beats implies paralysis of the vagus; increase above 180 beats, the combined effect of both causes.

The attack may be temporary, paroxysmal, or permanent. Temporary tachycardia may occur in any of the conditions mentioned and the pulse may in extreme cases reach 100–160, even 200 in the minute. But these cases, as stated, do not properly fall under this head. More properly tachycardia was the case of Tarchanoff's student who could increase the frequency of his own heart from 70 to 120 beats per minute.

Permanent tachycardia is an expression of organic disease, usually in the course of the vagus, and in the majority of cases at the origin of the vagus in the medullla. It is most frequent and most pronounced in Graves' disease, where it is associated with tremor, exophthalmos, and enlargement of the thyroid gland. The tachycardia is one of the cardinal signs of this disease, and is frequently the first sign. So much is this the fact that cases of tachycardia are often regarded as precursors, or more particularly as undeveloped cases, of Graves' disease. It is certain, however, that tachycardia may exist alone throughout the life of the individual; and it is also recognized that the disease occurs with greater frequency in the male sex than does Graves' disease, in which the female sex prepon-

derates. Graves' disease is regarded by many eminent authorities as, in its finality, a central bulbar lesion. Whether the tachycardia will be followed by other symptoms, tremor, exophthalmos, goitre, vaso-motor disturbance, etc., will depend upon the situation and extent of the lesion in the medulla.

One clear case described by Ohlenberg could be distinctly referred to implication of the vagus in the neck. In this case there was also mydriasis, moderate exophthalmos, with slight struma on the right side. Mention has been made elsewhere of the five cases collected by Proebsting, in which pressure upon the vagus was demonstrated at the autopsy, and of the observations of Riegel and Pelizäus in proof of the fact that caseous tuberculous glands often cause tachycardia with palpitation. Permanent tachycardia is also seen at times in pontine softenings associated with paralysis. Permanent tachycardia is, therefore, a very grave disease. But habitual tachycardia dependent upon toxic cause, alcohol, tobacco, coffee, tea, etc., or upon the circulation in the blood of toxins, as in uræmia, diabetes, gout, tuberculosis, carcinoma, will have a history and prognosis according to the nature of the cause.

Larcena divides the causes of tachycardia into the following groups, and makes thereby the best possible demonstration of the great variety of causes which may produce this symptom:

(1) Tachycardia in disease of the heart and blood-vessels. Under this head is included the increase in the action of the heart which occurs in overstrain, in the hypertrophy of growth, in acute and chronic myocarditis, in acute endocarditis, in valve diseases, in pericarditis, in angina pectoris, acute and chronic aortitis, in arteriosclerosis, and the affection of the heart which occurs in consequence of Bright's disease.

(2) Cases of febrile tachycardia.

(3) Tachycardia from peripheric compression—that is, of one or both trunks of the vagus, and from central compression of the nucleus of the vagus.

(4) Tachycardia from organic disease of the nervous system:

(*a*) Bulbar diseases, bulbar paralysis, softening in the medulla, bulbar meningitis;

(*b*) Certain medullary diseases, acute ascending paralysis, acute myelitis, progressive muscular atrophy, disseminated sclerosis, multiple sclerosis of the pyramids without lesion of the anterior horns, tabes dorsalis, syringomyelia;

(*c*) Organic disease of the peripheric nerves; degeneration of the vagus in tabes dorsalis, polyneuritis, beri-beri.

(5) Tachycardia in general diseases:

(*a*) Acute diseases, typhoid fever, diphtheria;

(*b*) Chronic diseases, tuberculosis, carcinoma, chlorosis, syphilis, chronic malaria, chronic rheumatism of the joints.

(*c*) Convalescence and exhaustion.

(6) Toxic tachycardias:

(*a*) From alcohol, coffee and tea;

(*b*) From drugs, digitalis and atropine.

(7) Reflex tachycardias from the brain, heart, lungs, stomach, liver, intestine, uterus, abdomen, bladder, prostate gland, brachial plexus.

(8) Tachycardia in neuroses: Graves' disease, hysteria, epilepsy, neurasthenia.

Paroxysmal Tachycardia.

Where the tachycardia occurs in attacks with more or less distinct intervals the condition is known as paroxysmal tachycardia. In the study of this form it is necessary here also first to exclude organic disease, especially of the heart or lungs, endo-, peri-, or myocarditis, pneumonia, œdema of the lungs, etc. Now it is a question if such an exclusion of organic disease is possible, as the tendency of the present day is to ascribe these cases to acute dilatation of the heart.

Paroxysmal tachycardia deserves the designation nervous only when it develops independently of any demonstrable disease. The tachycardia which occurs under the influence of poisoning by atropine or amyl nitrite, gives the clew to the proper understanding of the condition. In these cases the heart resumes its natural tone when the poisons or toxins are eliminated from the body. Paroxysmal tachycardia depends as a rule upon affection of the vagus, upon some lesion of the nerve centre, perhaps of the nature (molecular) of that assumed to exist in cases of pure epilepsy and pure migraine, conditions which also occur with paroxysmal explosion but which imply a continuous substratum. In a large percentage of cases the paroxysmal tachycardia is only the first link in a chain of disease processes which is finally fatal from paralysis of the heart.

As tachycardia, though it shows itself in the heart, is usually a disease of the nervous system, it is not surprising to know that it occurs often in connection with hysteria and neurasthenia, and that attacks are especially frequent under the use of agents which affect the nerves of the heart, alcohol, tobacco, etc., or that they occur in consequence of excesses which overstimulate or exhaust the nervous system, onanism, sexual excesses, coitus reservatus, etc. Most patients have reached the age of maturity. Tachycardia is rare under twenty, and children are almost never attacked. The increased

frequency of pulse, which is sometimes found in childhood, is rather the expression of a pavor nocturnus, epilepsy, malaria, or some cause which is obvious. The condition results, therefore, most frequently from long-continued overstrain or excessive irritation, the accumulated effects of which could only be experienced in middle life (Rosenfeld). Then, too, the affection is much more frequent in the male sex.

Tachycardia, like palpitation, may result from a direct reflex or toxic cause. The consideration of a case from this standpoint often leads the practitioner to a correct adaptation of therapy, but, in the present as yet confused state of knowledge of the complicated action of the cardiac nerves, it is needless to state that such a separation is not always possible. It must be admitted, also, that in many cases the action of these causes is conjoint.

Tachycardia is direct when it develops in consequence of some disease of the brain or spinal cord, or some affection which paralyzes the vagus or stimulates the accelerators at the origin, in the course, or at the termination, of these nerves. Thus Pepper saw tachycardia develop suddenly in a rapidly fatal case of myelitis. The cord showed on autopsy numerous small hemorrhages.

Reflex tachycardia develops from disease of some distant organ, and toxic tachycardia arises in consequence of the action of poisons, as of alcohol, tobacco, amyl nitrite, atropine, or other drugs, or of toxins which poison the nerve centres. Most cases resolve themselves finally as toxic, inasmuch as the prime cause of even sclerosis or of degeneration of the nerve centres (cells) must be ascribed to the effect of infections (micro-organisms) introduced, it may be, months or years before the tachycardia develops. A former mycosis is assumed to be the *fons et origo* of these cases. Hence where a cause is not demonstrable or discoverable, cryptogenetic is in all cases a better term than idiopathic or essential, for cases assumed to be essential sometimes turn out to be reflex, or to be due to actual organic disease of the heart. Thus Lainé reported a case of paroxysmal tachycardia which he had considered of the "essential" variety, but in which the tachycardia disappeared after the patient had passed a few calculi per urethram. Klemperer reported attacks of paroxysmal tachycardia of 200 beats in the minute in a case of dyspnœa in a woman with marked hyperacidity of the gastric juice, and Payne-Cotton observed a pulse of 232 beats in the minute, which was reduced to the normal after the discharge of a tapeworm—that is, the heart gradually returned to the normal under the use of digitalis. In these cases there was no record of the size of the heart during the attacks or previous to them, so that it was impossible to discern

their exact nature. Most cases of reflex tachycardia depend upon affection of the digestive tract. Fraentzel proved by a case which came to autopsy that paroxsymal tachycardia is sometimes a symptom of chronic myocarditis.

Symptoms.—The attack of paroxysmal tachycardia sets in, as a

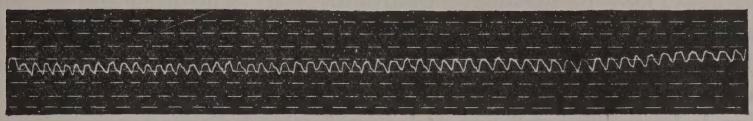

FIG. 19.—Paroxysmal Tachycardia. Radial pulse during the attack.

rule, suddenly, *i.e.*, without prodromes, in the midst of apparent health. The heart beating regularly, 60–80 strokes a minute, is suddenly increased to 120, 140, 160, or more. The increase is attended with, sometimes immediately preceded by, a sense of constriction in the chest, slight vertigo, or distinct malaise. There is often also a feeling of apprehension and anxiety, though never to the degree or

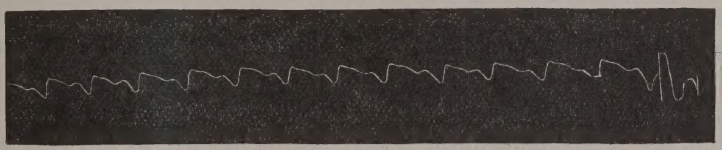

FIG. 20.—Paroxysmal Tachycardia. Radial pulse during intervals.

extent characteristic of common palpitation. Sometimes there is pain, more frequently a vague sense of distress or a feeling of oppression, in the precordial region. The breathing is usually slightly increased to 20–24, but respiration is in no way seriously affected or disturbed.

The prominent symptom is the increased frequency of the pulse

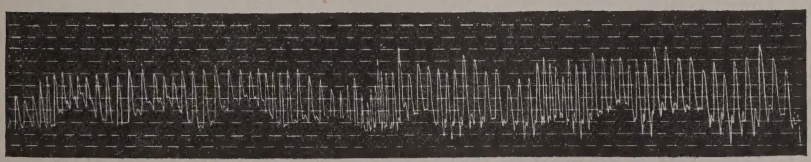

FIG. 21.—Paroxysmal Tachycardia. Pulsation of the jugular vein during the attack. (Martius.)

which has given the name to the condition. In many cases the beating of the pulse is so rapid that it may no longer be counted by the fingers at the wrist. The contractions of the heart are so frequent that many of the blood waves fail to reach the radial pulse. Schott

showed that the sphygmogram may appreciate a pulse at the radial, which the finger can no longer feel. The heart is then said to flutter or vibrate. In correspondence with the increase in frequency the force of the heart is reduced, so that the impact may be scarcely perceived or felt. Under this rapid action the difference in the sounds of the heart may be no longer distinguished. The first and second sounds are alike. The pause is not appreciated at all. The condition is that of the embryo heart, where the pulsations resemble the tick of a watch. In cases not so rapid it is seen that the sounds are pure and clear. There is no evidence of valve lesion.

Other than the rapid pulsation, the heart shows no sign of disease. The diameters are slightly increased during the attack, and the sounds are somewhat accentuated when the heart beats slow enough to recognize them, but there is no degeneration of muscle and no lesion of valve. These statements apply of course only to the cases of nervous tachycardia uncomplicated by organic disease.

The general evidence of stasis from defective circulation may be manifest in enlargement of the liver and hyperæmia of the lungs, which reveals itself in the râles heard over the postero-inferior aspect of the chest. The insufficiency of the heart is evidenced also in the pallor of the face, slight cyanosis about the lips, and anæmia of the brain, which in some cases amounts to syncope.

Thus the attack continues, it may be for but five to ten minutes, or for half an hour to an hour, or with remissions for several days. Bristowe reported a case which lasted continuously for five weeks. It is exactly in these long-continued cases that the frequency of the heart is increased to almost incalculable frequency (260–300).

During the whole period of attack the patient is for the most part speechless and helpless. He is unable to stand up or lie down, but sits dejected, the picture of silent suffering. It will be remembered, of course, that attacks vary in every degree of intensity and duration.

All at once the scene changes. The color returns to the face with an aspect of increasing comfort, the surface becomes warm or flushed, the pulse drops suddenly to the normal grade and sometimes sinks below it. In a case reported by Rosenfeld the heart beats fell suddenly from 180 to 80, and in the course of five minutes to 52, then to 40, and the bradycardia lasted from five to six days, as if the heart were recovering slowly from its excessive work. The change is announced at times by a sudden distention of the large arteries, as by the inundation of a tidal wave, followed by a number of almost imperceptible waves, whereupon the heart recovers its tone and the pulse becomes perfectly regular. A more or less profuse discharge

of urine and diaphoresis, or even a diarrhœa, may occur during the attack or mark the close. In some cases there is vertigo, in others actual vomiting, in still others the attack ends with sensations of peculiar character in the chest, which the patient often recognizes as the finishing stroke. The individual finds himself now as well as before. Nevertheless he may not be said to be in perfect health. The substratum upon which the disease is built remains. This condition may show itself in a series of light abortive attacks, or as a mere neurasthenia, or there may be other evidence of disease of the brain or of the distant organ which has excited the attack. The individual is rather in the condition which marks the intervals between explosions of migraine or epilepsy.

The attack may now repeat itself at any time, it may be in the course of the same day, or after several days or weeks or months. Rosenfeld recorded the case of a patient who had apparently entirely recovered, but suffered another attack after the lapse of fifteen years.

The *diagnosis* is readily established, as a rule, and the interest of the case turns upon the nature or the cause of the condition. The tachycardia must occur in paroxysms to constitute the distinct affection. The attack should show itself in the midst of apparent health, and be excited by trivial or undemonstrable cause. There should be absence of any evidence of organic disease of the heart. Stenosis at the mitral valve must especially be excluded. Attacks of diarrhœa, diuresis, or profuse sweating distinguish a purely nervous case.

Irritation of the sympathetic excites dilatation of the pupil, separation of the eyelids, protrusion of the bulb, pallor, and reduction of the temperature in the skin of the face. The finding of these symptoms in connection with tachycardia refers to affection of the sympathetic nerve (Oppenheim).

A chief distinction between a case of tachycardia from dilatation of the heart and tachycardia from other cause rests upon the fact that the dilated heart, in paroxysmal attacks, acts irregularly, whereas in true tachycardia the heart acts regularly. Cases of reflex tachycardia are distinguished by the fact that they are preceded by no signs of primary dilatation of the heart—that is, by no sinking of the blood pressure—and dilatation of the heart does not develop secondarily in these cases any more than in cases of tachycardia produced by anatomical lesion of the vagus nerves. The increase in the pulse frequency is in these cases but comparatively slight (Martius).

The pulse is increased in the highest degree in cases of acute dilatation of the heart. The tachycardia is the main symptom of this affection. Martius maintains that the term paroxysmal tachycardia

should be abolished altogether. The cause of acute dilatation of the heart remains obscure, but it will probably find its explanation in toxæmia. The subject has also been discussed in the section on Hypertrophy and Dilatation.

Fraentzel establishes his diagnosis as to the nerve affected *ex juvantibus*. Where the fault lies with the accelerators (sympathetic) the attack is cut short at once by morphine; where the fault is with the vagus by compression of the nerve trunk in the neck.

The *prognosis* in a purely nervous case *quoad vitam* is not bad, but is bad *quoad valetudinem*. However frequent the action of the heart, however profound the general distress, the disease almost never takes life. It is a matter of astonishment that the heart may beat with such rapidity so long without destroying itself. And in fact, in the course of time it does consume itself, but patients may live many years, suffering repeated attacks of tachycardia, in a condition of comparative comfort, and a fatal termination may usually be ascribed to complicating or causative organic disease. Nevertheless, Bouveret was able to find four cases of sudden death from heart failure in the course of an attack. If three animals be taken, all as nearly alike as possible, and the vagus nerves be cut in one so that the heart beats more quickly, and in another stimulated so that the heart beats more slowly, and the animals be all killed and the hearts exsected, it will be found that the heart which beats more quickly perishes the soonest, while the heart which beats more slowly outlives the heart which had not been experimented upon (Brunton).

So long as the arteries are filled, notwithstanding the tachycardia, the case is not grave, but when the heart's contraction is so rapid as to cease to fill the vessels, with evidence of cyanosis and stasis, the case assumes gravity. The presence of pain during the attack indicates a complication usually with angina pectoris, and greatly aggravates the prognosis.

The prognosis of permanent tachycardia is always serious, as it indicates in the majority of cases organic disease of the brain. The tachycardia may be, as stated, direct evidence of Graves' disease. The condition, however transitory, is never trivial. "Tachycardia can never be looked upon as a favorable sign, as it signifies arrest of the heart's action, and leaves to be feared the development of symptoms of weakness and exhaustion" (v. Basch).

Treatment.

Therapy resolves itself into treatment of the attack and of the interval, that is, into the attempt to cut short the paroxysm and to

prevent it altogether. In the attempt to abort the paroxysm avail is made of the well-known fact that compression or irritation of the vagus will retard the action of the heart. The Fakeers of India, it is said, can secure in this way such arrest of both circulation and respiration as to simulate death for a long time. A mechanical compression of the vagus in the neck succeeds in many cases in arresting the tachycardia. Bensel and Weidener established the diagnosis of a case as a transitory paresis of the vagus by cutting an attack short within two to five minutes by mechanical irritation of the vagus in the neck, and Czermak, Bensen, and Priesendörfer all record similar success with the same procedure. Nothnagel reported a case in which the patient succeeded in cutting short the attack by a prolonged and deep inspiration, a process which irritates the filaments of the vagus.

Rosenfeld reports that one of his cases made the accidental observation that the attack ceased immediately after a deep inspiration and strong and marked increase of intrathoracic pressure. Thereupon he tried this procedure in a case in which the paroxysm had lasted a quarter of an hour, and described it in detail as follows: The patient, a lady, laid herself horizontally in bed, raised the head a little, and pushed the feet against the bed. She then made a deep inspiration and pressed the lungs down with all her power by forcible closure of the glottis. The diaphragm was likewise fixed by energetic action of the abdominal walls; the arms were bent at the elbow and pressed against the chest and sides of the thorax, while contraction of the pectoral muscles drew the chest backward. Thus the contents of the thorax were fixed in every direction, from above, from below, and from the sides, for fifteen or twenty or more seconds. The immediate effect of this pressure was an increase in the pause of the heart, a momentary still-stand, followed by two or three strong and slow pulsations, and so the attack was ended.

"During pressure the face becomes bluish, the eyes are prominent, the lips and nose are cyanotic. But so soon as the pulse becomes normal, the usual color of the face returns." The patient feels somewhat wearied, but after remaining quiet for a few hours is able to assume her domestic duties. This patient subsequently tried the same procedure in a number of attacks and in every case with the same success. In the mean time, she has become so well acquainted with the method that she is able to execute it in a sitting posture. In three other cases which came under the observation of this author trial was made of the same mechanical method. "At first the experiment failed on account of the awkwardness of the patient, but now it succeeds in every attack."

Schott admits the value of this treatment, which is only a modification of massage in certain cases, but maintains that it cannot apply to all cases, especially to those which are of reflex origin, where the attack can be broken up only by removal of the cause.

Where the action of the heart is tumultuous, it may be stilled by the application of cold, as by an ice-bag or by cold compresses. Where the attack is of long duration the patient may wear the hollow helmet made of tin, the cavity of which is filled with cold water.

Narcotics, morphine, and atropine as a rule, have but little control over the paroxysms of tachycardia (vagus affection), though they may relieve the distress for a time by blunting the sensations, but usually at some cost to the subsequent comfort of the patient. Amyl nitrite and nitroglycerin succeed occasionally, but fail in most cases. Digitalis is of value only in cases of organic disease.

In the treatment of the interval the best results are obtained in cases of toxic and reflex origin, where bad habits are overcome or disease of distant organs can be cured. Thus new attacks may be prevented by regulation of the diet, avoidance of bodily and mental, especially emotional, strain, correction of constipation, reposition or curetting of a displaced or diseased uterus, cure of gout, etc.

The treatment of neurasthenia, hysteria, anæmia, and chlorosis will, when necessary, suggest itself. Finally, in a purely nervous tachycardia the influence of "suggestion," which is often ignored—though thoroughly utilized by the charlatan—will be brought to bear upon the appropriate case by the scientific physician, who will not hesitate to ransack every field of therapy to secure relief.

Bradycardia.

Bradycardia (*βραδύς*, slow, *καρδία*, heart), pulsus tardus, pulsus rarus, more properly araiocardia (*ἀραιός*, rare) or oligocardia (*ὀλίγος*, few). The term bradycardia, meaning only a slow heart, is really a misnomer, as it refers literally to the duration of an individual contraction. A real bradycardia is represented in the pulsus tardus in obstruction at the aortic valve. Araiocardia or oligocardia would better express the few beats in the minute as compared with the action of the normal heart.

Slow heart and slow pulse do not always correspond. In estimating the frequency of the action of the heart by the pulse it is necessary to distinguish between infrequent and incompetent contractions. Laennec said long ago: "We can distinguish two kinds of intermissions: the one *real*, consisting in an actual suspension of the heart's contractions; the other *false*, depending on contractions so

feeble as to be imperceptible or almost imperceptible to the touch in the arteries."

The pulse is slow naturally with some people, that is, bradycardia may be physiological. Tachycardia is the rule, but bradycardia has been found as an exception in early life, as early as at the age of five years. If there is any anomaly tachycardia is also rather the rule, but bradycardia is not uncommon in old age. Dehio attributes it to a loss of vagus inhibition, to correspond with the lessening demands of age. Heller mentions two people whose radial arteries did not beat oftener than 24 to 30 times a minute, and Balfour cites from Roux the case of an agriculturist who had gone through his military service without difficulty, who never had an attack either cardiac or cerebral, and was in the best of health, while his pulse rate was never over 34 to 40 per minute, and even a run of several minutes never raised it higher than from 50 to 55, and that only for a few seconds. Broadbent mentioned the case of an athletic young man who had a pulse of 36. Corvisart said that the pulse of Napoleon Bonaparte never ran over 40, even during the excitement of a battle. Slow pulse sometimes runs in families and the condition is compatible with perfect work on the part of the heart. Slow action of the heart is rather the rule in individuals of tall stature and small thorax. A slow pulse often goes with deliberation and good judgment.

How few shall be the number of beats to constitute a bradycardia? Most authorities consider anything below 60 a slow pulse, but even with an acceptance of this figure as final it is astonishing what differences are given by different observers. Thus Riegel found in 7,567 observations 1,041 cases of bradycardia, while Grob, in 3,578 observations, found but 82 cases. Authorities generally have noticed the great preponderance of men. Riegel found but half as many men as women. Grob's cases were all in men except four. In 100 observations Rosenbach found 4 cases of bradycardia, and of 100 cases of bradycardia but 6 were in women. This author, however, considers as bradycardia, without reference to any fixed number of beats, all cases in which the pulse falls, without cause, eight or ten beats below the natural frequency.

Bradycardia may be permanent, temporary or transitory, or paroxysmal.

Permanent bradycardia implies, as a rule, organic disease, usually of the nervous system or of the heart. Retardation of the action of the heart to 50 and less per minute is seen in certain diseases of the brain and cord and in conditions of stupor, in the entire absence of any arteriosclerosis, and typically in arteriosclerosis with thrombotic occlusion of the coronary arteries.

Permanent bradycardia is often seen in the course of chronic diseases of the spinal cord, myelitis, pachymeningitis, cerebro-spinal meningitis, and after injuries, hemorrhage, or concussion. Surgeons have long since recognized that fractures of the cervical vertebræ, especially of the fifth or sixth, frequently give rise to slow pulse. Gurlt says that fractures as low down as the seventh cervical vertebra may have this result, the pulse falling as low as even 20 per minute, and Charcot states that the retardation of the pulse is one of the most interesting facts of the symptomatology of cervical spinal lesion. Hutchinson says that unless injury to the spine is in the cervical region, no influence on the heart's action is ever observed (Balfour). Diseases of the brain also, meningitis, hydrocephalus, tumors, and abscesses, which make direct or indirect pressure upon the cord, may exercise the same influence on the pulse. Many individuals who show habitual bradycardia are found to be subjects of epilepsy or paralytic dementia.

Temporary bradycardia is not at all infrequent, and is observed in the course, or more commonly as the consequence, of many of the infections. Thus slow pulse is frequently found after typhoid fever and diphtheria, pneumonia, erysipelas, and rheumatism. Bradycardia is found in about twenty-five per cent. of puerperal women. Most of the cases observed by Riegel, referred to above, resulted from acute fevers. Baumgärtner reported a case in which the pulse sank to 25 late in the course of diphtheria, and Prentiss remarks upon the frequency with which the pulse is reduced to 26 per minute after typhoid fever. A slow pulse is found, as a rule, in connection with icterus, especially in the lighter catarrhal cases, and a slow, full, hard pulse often excites the first suspicion and leads up to the recognition of uræmia. Affections of the alimentary canal rank next in frequency to fevers in the production of a temporary bradycardia. A slow pulse is distinctly associated with constipation, and the sluggishness and tranquillity of the pulse stand often in marked contrast to the violent symptoms of occlusion of the intestine. Cold slows the action of the heart, and at 4° C. it stops beating. In the "sleep attacks" of hysteria the pulse may fall to 45 per minute (Briquet).

Paroxysmal bradycardia must occur, as the name implies, in attacks in the midst of perfect or apparent health, and to be of purely nervous origin the attack should cease more or less suddenly and repeat itself after some indefinite lapse of time. This bradycardia is often an effort of accommodation.

Bradycardia may be due to direct reflex or toxic cause, but the same qualifications apply to such divisions here as elsewhere in the

study of the neuroses, in that in so many cases two or more causes are conjoined. It is often difficult in the single case to isolate the predominant cause.

Most of the cases of permanent bradycardia are due, as stated, to organic disease, and most of these cases act directly upon the nerve centres or trunks. A typical example of bradycardia of brain origin was reported by Wreden in a case of abscess of the brain which finally perforated externally. The patient, a soldier, lay all the time in the left lateral position, crying on account of the severe headache. The pulse had become reduced to 15 and even 10 in the minute. In epilepsy, as stated, there is occasional marked reduction of the pulse, usually with arhythmia. Jacobi reported a case in which the pulse fell as low as 7 beats per minute.

A perfect example of the effect of pressure upon the nerve trunk was furnished by Czermak, who was able to arrest his own heart for several beats by pressure of the vagus against a bony outgrowth from one of his cervical vertebræ; Concato observed the same thing once in a patient.

Bradycardia from reflex action is assumed to exist in cases of affection of the abdominal organs. An important rôle is played here, however, by the distention of vessels with increase of blood pressure, which throws extra work upon the heart and leads up to hypertrophy and bradycardia. Thus Kisch found sometimes considerable retardation of the pulse during the process of the rest cure with over-feeding.

Toxic bradycardia finds its best illustration in the action of uræmia, icterus, and the toxins of the various infections (fevers). An outside illustration is found in the exquisite bradycardia produced by muscarine. That the extreme retardation of pulse following muscarine, which finally stops the heart in diastole, is not a paralysis is shown by the fact that mechanical irritation will excite a series of contractions. Cold-blooded animals, like the frog, in which the brain and cord still preserve their functions, continue to jump about and act in every way naturally for some time after the heart has entirely ceased to beat. Atropine is a perfect antidote, and the arrest induced by muscarine can be quickly relieved by the use of it. Lead also has a singular influence in retarding the action of the heart, and most cases of saturnism are marked by a slow pulse. Alcohol, tea, and coffee exercise this effect also with some people. The action of the heart becomes slow as well as feeble in all cases of nausea.

Disease of the heart itself produces or is attended with bradycardia only when the myocardium is involved. Aside from aortic stenosis where the bradycardia is the direct result of mechanical

obstruction, valvular lesions seldom show a slow pulse, and diseases of the myocardium are attended with bradycardia rather as the exception than the rule. Thus Quain reports of fifty-one cases of degeneration of the heart substance, only eight in which the pulse was slow. Occlusion of the coronary arteries is the most common cause of bradycardia in so far as the condition is dependent upon heart disease. Where the arteries are blocked suddenly, as by embolus or thrombus, the pulse is reduced at once, sometimes as low as 8 beats in the minute, and may not be again restored to its natural frequency even up to the time of death. More gradual occlusion, as in a more insidious process of thrombosis or progressive encroachment by aneurysm, may have ultimately the same effect. Pettus, of the U. S. Marine Hospital Service, reported the case of a fireman admitted to the hospital affected with fainting spells and shortness of breath. The cardiac rhythm was very slow but regular, numbering 24 beats to the minute. This bradycardia continued up to the time of death, thirty-two days after admission. Two months before, attacks of syncope, which had been diagnosticated as epilepsy, supervened upon any unusual exertion, and lasted two or three minutes. During the attack on the day of admission the beats fell to 14 per minute. The patient had had syphilis seven years before. Autopsy revealed an enlargement of the right auricle projecting outward from the right sinus of Valsalva, involving the orifice of the right coronary artery, which was completely blocked by a laminated blood clot.

An explanation of the action of occlusion of the coronary arteries is found in irritation of the terminal filaments of the vagus in the heart. Failure of nutrition is probably the more correct interpretation, as when entirely removed from the body and disconnected with all nerve supply, the heart beats slower and slower until it finally ceases altogether. Even then it may be restored to movement for a time by mechanical or other stimulation. This movement also becomes more and more sluggish, until finally there is no response to any stimulus.

Retardation in the action of the heart from occlusion of the coronary arteries has been so uniformly observed, as the result of experiment, as to have led to the erroneous belief that most cases of bradycardia depend upon this cause, and the influence of occlusion of the coronary arteries has therefore been exaggerated. Thus Prentiss reported the case of a man, aged fifty-three, having a pulse ranging from 11 to 40 per minute for over two years, who was subject to frequent attacks of syncope. No disease of the heart or other organs could be discovered, and the diagnosis was generally assumed to be a coronary sclerosis. In the later stages of the disease the patient became deli-

rious, showing the mania of terror. On the last occasion he ran about the house exclaiming, Murder! Fire! until the neighborhood was roused. He was induced finally to lie down, when his wife left him for a few moments, and on returning found him dead. The autopsy by Edes showed the heart normal. Neither the aorta nor the coronary arteries were atheromatous. The recurrent laryngeal nerve showed a number of fusiform enlargements which were supposed to have caused the slow pulse and the other symptoms, as one of them was on a cord going downward to form the œsophageal plexus, while others were on the bend of the recurrent laryngeal just below the artery, at the point where the recurrent laryngeal gives off branches which connect with the cardiac plexuses.

Pericarditis shows bradycardia at times from increase of the peripheric resistance, which retards the action of the heart, or from pressure of the exudation upon the inhibitory fibres at their entrance into the pericardium (Rosenbach).

Prentiss analyzed all the cases reported in the library of the Surgeon-General's office of the U. S. Army. The whole number was 89. The causes assigned were stated as follows: Disease of the brain, 8 cases; disease or injury of cervical vertebræ, 11 cases; epileptic convulsions, 7 cases; heart disease, 9 cases; ossification of aortic valves and coronary arteries, 3 cases; starvation and exhaustion, loss of rest, convalescence, 3 cases; lead poison, nervous shock, fever, 3 cases; poison by salt fish, 1 case; cholera morbus, 3 cases; acute febrile condition, 4 cases; pericarditis, 2 cases; cerebral convulsions, 2 cases; rheumatism, 1 case; sunstroke, 1 case; cause not given, 35 cases. The slowest pulse recorded was in Case 79, 3 per minute for several hours. In Case 53 it fell to 4 per minute for four minutes during an attack of syncope, and once it did not beat for thirty-five seconds, and again for twenty-five seconds. The next slowest pulse was 7 per minute, in Case 36. In Case 3 it fell to 9 per minute.

It is evident that bradycardia depends in the main upon some organic disease. It is, therefore, much less frequently a neurosis than tachycardia. It is, indeed, a question if the causes which are called toxic do not in all cases act upon the muscle protoplasm itself. The retardation which occurs in consequence of arrest of respiration, as in holding the breath, is cited as positive proof of the influence of irritation of the vagus and justification of the consideration of bradycardia as a neurosis. But even this simple evidence admits of another interpretation in the slowing of the muscular contractions under defective oxygenation. For the heart becomes irregular as well as slow, arhythmic as well as retarded, under suspension of breathing. The heart out of the body beats longest in pure

oxygen and stops very soon in an atmosphere of chlorine or of carbonic dioxide.

The heart struggling under the action of poisons, or endeavoring to adjust its action to various demands, is much more apt to have its beats quickened than slowed; therefore paroxysmal bradycardia is not so frequent as paroxysmal tachycardia, considering as a true bradycardia only those cases in which the pulse rate is reduced to 60 and less.

Symptoms.

As most cases of bradycardia depend upon organic disease, and the slow pulse is associated with other more obtrusive signs, we shall consider here only those cases in which the bradycardia bears the aspect of a more or less pure neurosis. This qualification applies more especially to paroxysmal bradycardia, where the attack, as stated, sets in more or less suddenly in the midst of apparent health, and lasting an indefinite time, associated for the most part with other nervous symptoms, disappears in the same way. In exceptional cases the attack is preceded by malaise and a feeling of weariness or exhaustion. Not infrequently the attack begins with a violent palpitation, with a sense of oppression and constriction, sometimes with dyspnœa, which may increase to suffocation. In other cases there are sensations of distress on the part of the stomach, nausea, retching, vomiting, with subsequent collapse. In grave cases the anxiety deepens into a sense of impending death. The extremities become cold, the surface is covered with a clammy sweat. The patient falls into convulsions or syncope. In lighter cases there is a feeling of unrest and anxiety, sometimes attended by sensations of distress in the abdomen, distention, tormina, and tenesmus, in rarer cases by dysuria. In others there may be evidence of implication of the nerves of the larynx, shown by hoarseness, aphonia, or stridulous breathing. In still other cases the attack is associated distinctly with excruciating and agonizing pain, confined to the region of the heart or radiating thence to the shoulder and the arm. These cases are distinctly complicated with or produced by the same cause as angina pectoris. The relation to epilepsy has been already mentioned. As a rule the attack itself is free from convulsions, but in some cases an explosion of epilepsy occurs, when the pulse reaches its lowest rate. The epileptiform attack may be here a mere consequence of anæmia of the brain, but in many cases both the bradycardia and the epilepsy are due to a common cause, that is, to change in the central nervous system.

Strübing relates the history of a case of his own in connection

with five of Mosler. The case of Strübing was interesting as it occurred in youth, which is rare. A boy, aged fifteen, had often suffered dyspnœa after effort. One day, after he had taken a long run, there occurred several attacks of unconsciouness with vomiting. The heart dulness was markedly increased, extending over the anterior axillary line up to the right border of the sternum. The heart beats were reduced to 16 to 18 per minute. The sounds were faint and dull; there were no murmurs. In the course of several subsequent attacks the pulse sank to 14 and even 12 in the minute, so that death was expected. But in the course of three weeks the heart's action increased to from 40 to 44 and the patient was free of all difficulty. Hereupon a new effort brought about the same condition, which after a temporary improvement was followed by death in the course of a year. A post-mortem was not permitted. In this case rest of body and mind increased the strength of the heart without increasing its frequency, but the increase in strength was attended with comfort. As soon as the pulse was excited by psychical disturbances or by fever, disturbance of compensation at once set in. Strübing warns therefore against the use of excitants and recognizes the value of rest, with careful gymnastics later.

The pulse itself may be full and firm with the heart muscle sound

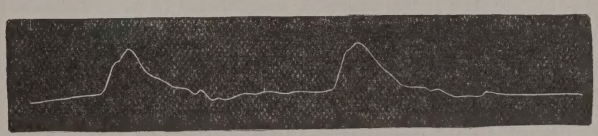

Fig. 22.—The Pulse of Bradycardia.

and strong, or faint and feeble in the presence of degenerative change. Fig. 22 shows a tracing from a case of purely nervous disturbance.

Diagnosis.

The diagnosis is determined by the evidence furnished by the pulse alone, and the point of practical interest turns upon the detection of the cause. It may be accepted as a rule, that permanent bradycardia when at all pronounced or excessive depends upon organic disease, usually of the brain or cord. The cause of temporary bradycardia may be discovered in the history of the case, in some previous infection (diphtheria), in the puerperium, in nicotine poisoning, etc. Most cases of reflex bradycardia are due to affection of the gastro-intestinal tract, especially of the abdominal viscera.

The main factor in diagnosis is the determination whether the

bradycardia is due to weakness of the heart muscle or to disturbance of innervation. Where the muscle is weak the pulse is small. Where the bradycardia depends upon disturbance of respiration, the pulse, though slow, is full and firm. It is "the pulsus tardus with the powerful arterial wave" (Rosenbach). The bradycardia of the hysterical attack is distinguished from the pure paroxysmal bradycardia by the fact that hysteria has forerunners in emotional disturbance, insomnia, irritability, dyspepsia, etc., that laughing and crying seizures occur during the attack and in the intervals, that other evidence of hysteria may be present, and that the increase of pulse previous to the bradycardia rarely reaches 130 beats per minute. Moreover, the attack never ceases so suddenly in hysteria.

Prognosis.

In a general way, the prognosis of bradycardia is more unfavorable than that of tachycardia, but a temporary or paroxysmal bradycardia, depending by no means of necessity upon organic disease, may have a favorable prognosis. Bradycardia from the infections and during convalescence usually disappears in the course of a few weeks or months. The bradycardia of the puerperium must be distinguished from that of Bright's disease, which has a more unfavorable prognosis. Any associate irregularity, arhythmia, aggravates the prognosis according to its degree. Any diminution in the force of the heart makes the prognosis more grave, as it soon leads up to the signs of anæmia of the brain, cyanosis, and stasis. The prognosis becomes much more unfavorable when the bradycardia is associated with any disturbance, especially with an increased frequency of respiration, and the presence of râles or crepitus in the lungs makes the outlook very bad. Any complication with organic heart disease intensely aggravates the prognosis. Thus a marked retardation of the pulsations of the heart is to be regarded as an injurious complication in insufficiency of the aortic valves especially, because it increases the duration of the diastole, and thus favors the reflux of the blood into the left ventricle. It may also be regarded as a serious complication of mitral insufficiency, as the rapidity of the systole contributes essentially to the fact that the aortic system is comparatively better filled than the left auricle (v. Basch). The more slow the pulse rate, the more rare are murmurs in the heart or vessels. At the rate of 40 beats accidental murmurs are never to be heard, though there may be a splitting of the first sound and a marked accentuation of the second. Most weight is to be laid upon the splitting of the first sound, as when pronounced it indicates certain weakness of the heart muscle.

If there is fever and if the fever is out of proportion to the activity of the heart, if the face is cyanotic, or if the body is covered with sweat, if the mind is obtunded, or if there is delirium, the prognosis is very bad. A retardation of the pulse in severe diabetes is usually connected with albuminuria and heart weakness, and is thus an extremely unfavorable sign. The most favorable prognosis is offered in cases of poisoning by nicotine.

Treatment.

The treatment of permanent bradycardia will depend altogether upon the cause, whether disease of the brain or cord, arteriosclerosis, or aneurysm.

Reflex bradycardia may be relieved by the removal of some distant affection of the abdominal organs, as by the treatment of gastric catarrh or atonic dyspepsia, by relief of constipation, the discharge of intestinal parasites, etc. Chronic conditions of disease of the abdominal and pelvic organs, associated especially with constipation and flatulence, may be further relieved, and attacks of bradycardia prevented by systematic massage and galvanization of the abdominal walls. So long as the heart feeds the brain and the body by the force of its contractions, in spite of their infrequency, little demand is made for treatment. But so soon as evidence of anæmia of the brain or stasis supervenes, that is, so soon as the pulse becomes excessively slow or weak, resort must be had to stimulants. Great care, however, must be exercised in the use of powerful stimulants like digitalis, nitroglycerin, strophanthus, and sparteine, and these agents should be used at first in small and tentative doses. But where the heart failure is pronounced, as manifest by œdema of the lungs or cyanosis, the stronger and more diffusible heart stimulants must be used at once. Thus the sodium benzoate of caffeine, grs. iii.–v., may be administered every hour or two. This remedy may also be employed in solution subcutaneously, or resort may be had to hypodermic injections of the ethereal solution of camphor (1–10). An attempt may be made to stimulate the heart with alcohol, especially in the form of brandy. A cup of black coffee containing a teaspoonful or two of brandy is a powerful stimulant to the heart.

An attack of paroxysmal bradycardia may sometimes be cut short by the relief of flatulence, as by the ingestion of the oil of turpentine, gtt. x.–xv. in a wineglass of milk, or by a teaspoonful to a tablespoonful of the milk of asafœtida, or a teaspoonful or two of the tincture of valerian, or sulphuric ether, gtt. xx.–xxx., or half a teaspoonful to a teaspoonful of Hoffmann's anodyne.

Finally, appeal may be made to morphine, which is of especial

value in quieting associate nervous distress and anxiety. Morphine is, therefore, particularly adapted to the purely nervous cases of reflex or paroxysmal bradycardia. It should be used with much caution in cases of organic disease and in the presence of heart failure, and great care must be taken not to give it *in extremis*, that the drug be not blamed for the death. Sometimes the extract of Indian hemp exercises a very favorable effect.

The action of atropine in so quickly neutralizing the effect of depressing heart poisons may justify the use of this remedy in tentative dose. Atropine, gtt. iii.–v. of the one-grain to the one-ounce solution, may be given with or without morphine.

During the attack the extremities must be kept warm. Sometimes anæmia of the brain is prevented by laying the head low, and sometimes it is prevented by bandaging the extremities, a process which also retains heat.

During the interval between attacks, the heart and the nervous system may be braced by the systematic use of strychnine, and especially by baths and by judicious gradation of exercise in the open air.

Arhythmia.

Arhythmia (written also arrhythmia; from *α*, priv. and *ῥυθμός*, rhythm), pulsus irregularis, inæqualis, is a disturbance in the regularity or rhythm of the action of the heart. The neuroses hitherto described have had reference to disturbance of the frequency (tachycardia, bradycardia) or force (neurasthenia, palpitation) of the action of the heart, but abnormalities may occur also in the succession of the heart beats, which may be, as stated, irregular or unequal. These conditions of very varied character may be described under the term arhythmia. The heart in its work must propel the blood against the resistance of a constant pressure of blood in the vessels. This work is estimated at one hundred and twenty-four foot-tons in twenty-four hours (Houghton), the work of the left ventricle being about three times as great as that of the right. As the heart must be continually filled and emptied, the work must be done *saltatim*, and this result is secured by rhythmical contraction. The rhythmical motion begins in the auricles at the orifice of the emptying veins, and is propagated by means of directly connecting fibres which pass from the auricle to the ventricle, by undulatory movement, to the ventricles. The pause between the contraction of the auricles and the ventricles is therefore apparent and not real. With 65 pulsations to the minute, auricular contraction occupies 0.177 second, ventricular 0.34 second, the diastole 0.4 second; but

with very rapid pulsations the ventricular systole occupies only 0.199 second (Landois). In a heart which is beating very rapidly all these periods of time are shortened, but the shortening occurs mainly at the expense of the diastole (Donders, Chauveau, Burdon Sanderson). The disturbance in the rhythmic motion of the auricles and of the ventricles may be studied in the act of dissolution, when it is seen that the auricles continue to contract a short time after the ventricles have ceased. As the heart beats more slowly, there is a marked interval between the auricular and ventricular contraction, and in many animals there will be two or three auricular systoles to one ventricular (Thompson).

The heart entirely deprived of blood will still beat rhythmically outside of the body, but ligation of one coronary artery in a dog will, in two minutes, cause the regular cardiac contractions to give place to fibrillar twitching, and that ventricle is first and chiefly affected whose coronary has been ligated (Sée).

The cerebro-spinal system has most to do with the regulation of the force and frequency of the heart's action, but the regulation of the rhythm stands in closer relation to the sympathetic nervous system, and especially to the ganglia in the substance of the heart. The experiments of Wooldridge demonstrated the sensitive nature of the nerves which constitute the plexus at the bulb. Irritation of the central trunk of the ventricular nerves, which failed to excite motion, produces changes in the frequency of the pulse, alterations of pulsation in the arterial system. The rhythmic motion of the heart is due, as stated, to automatism of the heart muscle itself, and not to automatic action of the nerve apparatus. The ganglia have to do with regulation of the heart only indirectly by the perception of impressions in the sphere of sensation.

Ample provision is made for the nutrition of the heart ganglia. They rest at places where there exist numerous anastomoses between the terminations of the coronary vessels and branches of the anterior and posterior mediastinal arteries. The heart ganglia are therefore not nourished exclusively by branches of the arteries of the heart. Every ganglion is surrounded by a thick network of small blood-vessels, and numerous branches penetrate into its interior. Thus the heart ganglia are in no sense degraded by being denied the office of presiding over the motion of the heart; they have the higher one of perceiving the first influence of failing nutrition or toxic impression.

Notwithstanding its continuous work in supplying the varying wants of the body, the rhythm of the heart is so regular as to have at all times excited admiration. But very close examination will often detect variations which are not otherwise apparent. From

the investigations of Hüsler, in one hundred persons, healthy, sick, and convalescent, made with every precaution, it was seen that the regularity of the pulse rhythm is only relative. The healthy heart shows in its rhythm remarkable differences which cannot be explained

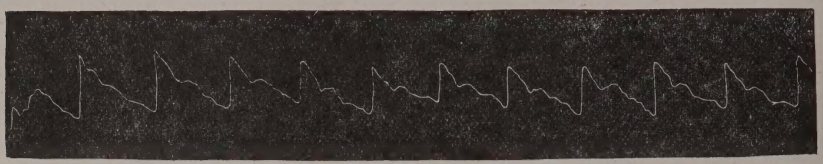

FIG. 23.—Normal Pulse Curve showing the Elevation of Systolic Impulse and the Arterial Recoil.

at the present time. These differences are so great as to leave no limit for a physiological pulse rhythm. It is a curious fact, however, that aside from these variations in health there is a remarkable tendency to the preservation of rhythm, as even grave diseases have usually no influence in disturbing the rhythm of the valve apparatus. Hüsler found that for the production of arhythmia there must be

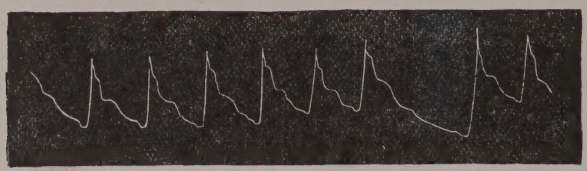

FIG. 24.—Pulse Intermittent Every Seventh Beat. (Pond.)

present some as yet unknown toxin which distinctly disturbs the mechanism of the heart's action. The rhythm of the heart is naturally changed in some people, and slight deviations are not incompatible with regular work. Thus a certain degree of arhythmia may be normal. These irregularities are noticed more frequently in childhood and then more especially in sleep. Under

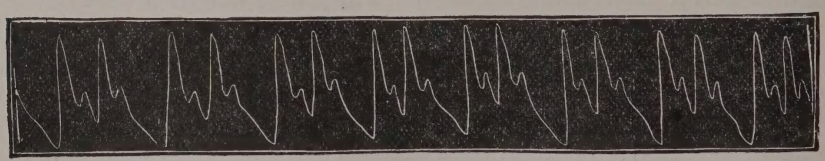

FIG. 25.—Pulsus Bigeminus.

bodily excitement or the presence of fever with increase of frequency, the irregularity disappears. Any irregularity of this kind in adults which disappears entirely under bodily or mental excitement has no pathological significance. But any irregularity which

is increased by excitement, or any marked irregularity at any time, especially if associated with any kind of distress or pain, is always a sign of gravity.

Arhythmia, as will be seen, may be of both muscular and nervous origin. Great stress is usually laid upon arhythmia as evidence

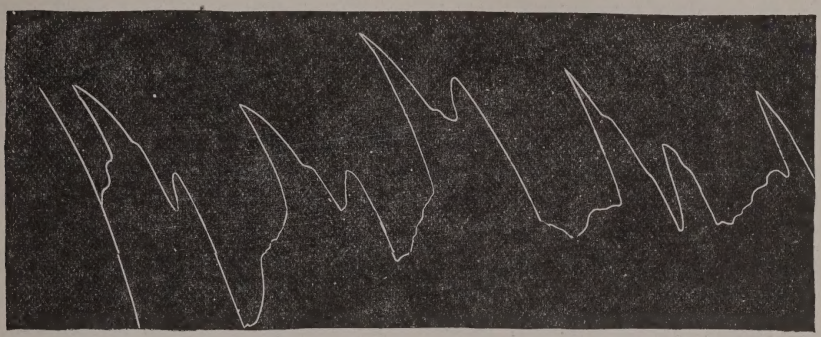

FIG. 26.—Pulsus Bigeminus Alternans. (Eichhorst.)

of myocarditis. It is interesting to know that the rhythm of the heart may remain normal even with severe disease of the heart muscle, and that even a long-continued irregular pulse may be independent of myocarditis, and may be, as stated, especially in young persons, a condition without any special gravity.

The most distinct irregularity is the loss of a beat, which is distinguished as an intermittence. In cases of intermittence of the pulse, it is necessary to ascertain whether it be real or false, that is, whether it is an expression of an insufficiency or of a real ventricular halt. Any doubt is determined by direct auscultation of the heart.

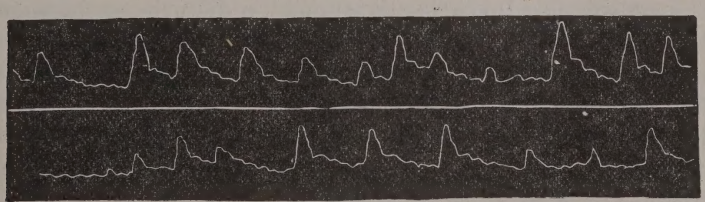

FIG. 27.—Forms of Arhythmia. (Fothergill.)

Hochhaus and Quincke speak of what they call "frustrate contractions" which show themselves in a certain rhythm; for instance, the second, third, even up to the tenth or fifteenth, contraction may be frustrated. In other cases two such frustrated contractions occur successively or they may be irregular for a long time and then show themselves in rapid succession.

According to the subjective statements of patients, this symptom shows itself quite frequently in the very first stages of heart weakness in middle life and in more advanced age. It is probable also that such frustrate contractions may occur now and then in the perfectly healthy heart under pure nervous disturbance. They have, therefore, no really definite meaning in prognosis.

Sometimes intermittence is incomplete. The action of the heart is distinguished by successions of strong and weak beats. When strong and weak beats alternate regularly with each other the condition is distinguished as the pulsus alternans. The rhythm is more distinctly disturbed when the successive strokes are closer or farther apart, or when the pause or interval between the strokes is longer. Two strokes in quick succession, followed by a longer pause, constitute what is called the pulsus bigeminus. Three quick strokes with a longer pause constitute the pulsus trigeminus. Combinations of these conditions may occur, as in the case of the pulsus bigeminus alternans, which consists of two contractions in quick succession, one being stronger than the other, followed by an abnormal pause.

Where the irregularity of the heart is always the same, that is, where a distinct method is observed, the condition is known as allorrhythmia (ἄλλος, another). In this condition a certain regularity can be detected in the apparent irregularity. It is only in the absence of any such method, or where the regularity is entirely anomalous, that a pure arhythmia may be said to exist. When, in addition to this state, the heart's action is tumultuous, the condition is known as a delirium cordis; or when the heart, besides being arhythmic, is rapid and weak and shows in its contractions only a series of vibrations, the condition is called a delirium or tremor cordis.

The pulsus paradoxus is not so much a disturbance in the action of the heart as of the pulse. In this condition the pulse instead of becoming stronger becomes weaker during inspiration. The rhythm of the heart may be in no way disturbed, but the contraction is too feeble to propel the blood with sufficient force through the vessels of the thorax, the distention of which by inspiration interrupts, *i.e.*, holds back, or arrests the blood wave.

Arhythmia may be due to direct, reflex, or toxic cause, sometimes to the combined operation of any two of these causes.

Under the *direct causes* are included all the diseases of the brain and spinal cord which may directly or indirectly affect the accelerator or pneumogastric nerves. Meningitis in its various forms often thus shows a typical arhythmia. Pressure upon these nerves in their course may also have the same result, so that arhythmia often exists

in connection with neurasthenia, palpitation, tachycardia, bradycardia, and angina pectoris, and the causes which produce any of these affections may operate also to produce arhythmia. Neoplasms in the neck or in the mediastinum, hyperplasiæ of the thymus or of the thyroid, aneurysm of the basilar artery or of the aorta, may act in this way.

Among the direct causes may be included also affections of the heart, which interfere with its action (peri- and endocarditis, myocarditis, valve lesions, atheroma, etc.), and of the blood-vessels (arteriosclerosis), which throw extra work upon the heart and thus derange the circulation; finally conditions of the blood itself, especially impoverishments and degradations, anæmia (hemorrhage), chlorosis, leucocythæmia, which markedly interfere with the nutrition of the heart. External pressure upon the heart causes it to beat more vigorously or elicits distinct arhythmia. Thus a distended stomach in flatulent dyspepsia may make pressure upon the heart to such a degree as to lead to collapse and syncope. Arhythmia always occurs before death,

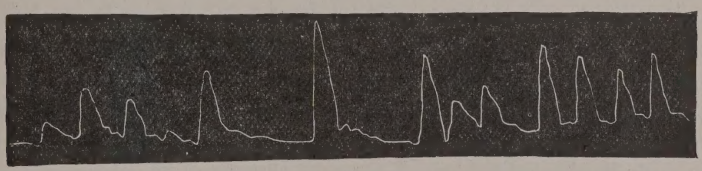

Fig. 28.—Ante-mortem (Preagonal) Pulse.

and the physician bases his estimate of the duration of life at the close on the irregularity and inequality as well as on the frequency and feebleness of the pulse.

Reflex causes are the diseases of distant organs, especially of the abdominal organs. Thus the heart may be rendered weak or its movements be absolutely arrested by blows on the stomach, operations on the uterus, etc. Brown-Séquard, in his experiments, saw arrest of the heart after removal of the semilunar ganglia, but if the vagus had been previously cut this arrest did not occur. Lancereaux noticed especially the influence of injuries in producing sudden death by reflex arrest of the action of the heart, and Golz reported eight cases of laparotomy in which, after operation, irritation of the intestine and of the internal pelvic organs exercised a reflex inhibition on the activity of the heart, and chiefly on the respiration. Sustained chloroform narcosis inhibits the reflex effect of operations in the abdomen upon the heart and respiration to some extent. The reflex irritability of the nerves of the heart and respiration is different in different individuals, but a weak mechanical irritation suffices to let

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loose an inhibitory reflex. In accord with what is known of the changes in nerve cells in exhaustion it is noticed that the effect is produced easier when the irritation is frequently repeated. Pain from any cause, no matter how or where produced, may derange the rhythm of the heart or even actually arrest its movements. Thus arhythmia is nearly always observed in association with the excruciating pains attending the passage of gall and kidney stones, and sometimes with the terrible torture of angina pectoris.

The *toxic causes* may act upon either the heart muscle or the heart nerves. Infections in fulminating form (scarlatina, cerebro-spinal meningitis, cerebral rheumatism), which destroy life in the course of a few hours or days, act by poisoning the heart muscle; the toxins of other infections (diphtheria, pneumonia) show their effect later in paralyzing the heart muscle.

Typical arhythmia is produced by the action of the heart poisons, especially by muscarine. After the administration of muscarine, there occurs, as stated previously, a marked retardation in the action of the heart. This period may last many minutes, and upon it supervenes a second period during which the heart beats irregularly and the pulse is arhythmic. v. Basch attributes the period of arhythmia to the combat with each other of two influences, one of which endeavors to retard the action of the heart, the other to increase it. During the first part of the effect of muscarine the first influence predominates. Irritation of the accelerator in the stage of arhythmia checks it and restores the normal rhythm. That the excito-motor fibres resist paralysis longer than the inhibitory vagus fibres is a fact which has been demonstrated by Truhart and Schmiedeberg in experiments on frogs, in which after removal of the inhibitory influence of the vagus, a rapid increase in the action of the heart was induced by long electrization of the peripheric vagi. Strophanthus, digitalis, chloral hydrate, aconite, veratrum viride, may all act in the same way as heart poisons. Alcohol in long-continued action produces arhythmia through organic disease and exhaustion, but tobacco exercises its influence more directly. Nicotine in very small dose at first retards the action of the heart (Truhart, Schmiedeberg) and in slight degree increases its force (Sée). In larger quantity it may lead to arrest in diastole. After a certain period, which is very short when large doses have been given, the heart's action is increased in frequency and then again retarded, whereupon it becomes irregular and interrupted, and finally so feeble as to be scarcely perceptible. The retardation of the pulse frequency may not be referred to central influences because it occurs after previous section of the vagus. It must depend upon irritation of the peripheric vagus filaments in the heart

or upon the heart muscle itself. This view is confirmed by the fact that after the injection of curare (Rosenthal) or atropine (Schmiedeberg), agents which paralyze the terminal filaments of the vagus in the heart, retardation of the pulse does not follow small doses of nicotine (Dornblüth). De Caisne found among eighty-eight smokers whom he examined intermittent action of the heart twenty-four times. Richardson maintains that any intermittent action of the pulse is intensified by the use of tobacco, and Liebermeister comments upon the fact that an intermittent action having been once produced by excessive smoking is liable to recur after the use of only one or two cigars a day.

Symptoms.

The symptom which gives the name to the condition is the disturbance in the regular rhythm of the pulse, which may present itself, as stated, as irregularities in force, space, and time. Many cases can be distinguished as true allorrhythmia, where the pulse intermits every sixth, tenth, or twelfth beat. The interruption or irregularity is for the most part unobserved by the patient, whose attention is usually called to it for the first time by the physician. Sometimes these intermissions are observed only when provoked by a definite exciting cause, as by the use of alcohol or tobacco, more especially by the ingestion of heavy meals, after a dose of chloral, or in the course of the use of digitalis, though more frequently an intermittence is made to disappear under digitalis. Sometimes the intermission or interruption is attended with a disagreeable sensation of which the patient is conscious. Where a number of beats are lost the patient experiences a condition of anxiety, which may border upon the unspeakable terrors of an angina. Romberg relates the case of a man, aged thirty-six, in whom there was a complete intermission of five or six beats. "The aspect of the patient showed that something dreadful was going on within him. He sat as if thunderstruck (attonitus), speechless, motionless, his eyes wide open, his consciousness unimpaired." The attack, which was caused by some emotional disturbance, was preceded by a presentiment, and was attended by violent pain in both sides of the thorax which extended to the nape of the neck and the head. After consultation with Skoda and Rokitansky, a diagnosis of tumor of the cardiac nerves was established. The attacks increased in severity and the patient finally died in stupor. It was found upon post-mortem that the cardiacus magnus nerve was woven into a black knot the size of a hazelnut; the left vagus was involved in an underlying nodulated dark-blue lymphatic gland, the phrenic nerve was also embraced in a diseased

gland. Irritation of the inhibitory fibres of the vagus was the cause of the long intermission. In other cases the arhythmia occurs in connection with the tumultuous contractions of palpitation, and the patient may experience the peculiar distress or actual pain which shows itself in this condition. Sometimes there is distinct evidence of stasis in hyperæmia of the lungs, cyanosis, and dropsy.

The associate symptoms will depend upon the cause of the condition. Thus there may be evidence of some affection of the brain (meningitis, tumor), or of the heart (myocarditis, arteriosclerosis), or of the blood itself (anæmia, chlorosis), of the action of toxins (infections, tobacco, etc.). The attacks of arhythmia which occur in connection with tabes dorsalis have been distinguished as heart crises. Like the other crises (laryngeal, gastric, nephritic) of this disease, they occur without apparent cause, are attended with excruciating pain, and often with extreme anxiety, sometimes with actual syncope.

Prognosis.

The prognosis depends entirely upon the cause. The arhythmia of young people, as stated, may have no special significance, but in a general way arhythmia is a sign of more grave import than palpitation. Aside from the cause, the gravity of an individual case as indicated by this single symptom depends upon the fact whether or not the arhythmia interferes with the blood supply. An arhythmia which attends or results from insufficiency from whatever cause is always an index of gravity. An arhythmia which disappears upon effort or excitement may be of little significance, but an arhythmia which occurs under a slight bodily effort, as by merely rising to the sitting or standing posture, or by walking about a room, indicates an insufficient response to the appeal to the heart for greater blood supply, and may be an early sign, therefore, of heart failure. The most favorable prognosis is offered in the cases of nervous arhythmia produced by the action of toxins, including alcohol, tobacco, coffee, tea, etc.

Treatment.

The treatment will address itself to the cause, myocarditis, arteriosclerosis, anæmia, dyspepsia, etc. The various toxins may be more readily eliminated through the kidneys and skin by diaphoretics (hot baths), diuretics, such as moderate doses of calomel, diuretin, or by heart tonics, such as strychnine or digitalis, which may drive the blood more rapidly through the kidneys. Sometimes the draining of dropsical legs by punctures or by a cannula relieves the

heart, so that an arhythmia of considerable duration may at once entirely disappear. Nitroglycerin, one or two drops of a one-per-cent. solution, often proves of great value in these cases, both by supporting the heart and by dilating the distant capillaries. A threatened collapse is often bridged over in this way, after which the patient, even with organic disease, may survive for years. The application of cold to the heart stills tumultuous action and stimulates the accelerator nerves. Extreme cases of arhythmia may be benefited most by the use of the general hot bath, while light cases may be relieved by the stimulating effect of the colder bath. The bath by stimulating the heart favors the elimination of toxins. The subcutaneous transfusion of the physiological salt solution (0.6 per cent.) one-half to one pint, by feeding the blood supply, may quickly discharge an arhythmia of long duration.

In extreme nervous arhythmia with distress and danger, morphine is the remedy above all others on account of its quick action. It is especially indicated in sudden arrest of respiration and circulation during the narcosis of chloroform, in case of drowning or of an individual suffocated by smoke. Morphine is also indicated in a prophylactic way in patients affected with tumors, especially in the neighborhood of the vagus (Lancereaux). The subcutaneous use of morphine preceding a severe or extensive surgical operation protects the heart against excessive reflex inhibition.

An arhythmia of purely nervous origin disappears readily, as a rule, with removal of the cause (relief of anæmia, the restoration of menses, abstention from tobacco, etc.), or may be made to disappear by the simpler heart tonics, the tincture of nux vomica, with regular habits, baths with massage, good food, and fresh-air exercise.

Angina Pectoris.

Angina pectoris (Heberden, 1768, angere, to bind, to strangle), stenocardia (Breva), sternalgia (Goode), syncope anginosa (Parry), neuralgia plexus cardiaci (Romberg); Breast-pang, German, Brustbräune—is an affection of the heart characterized by paroxysms of agonizing pain of sudden occurrence which irradiate usually to the shoulder and left arm, attended with an awful sense of impending death, caused by sclerosis of the coronary arteries or atheroma at or about the aortic orifice.

Two varieties of angina are distinguished, viz., true angina, dependent upon organic change, and false (pseudo-) angina, which is more distinctly a neurosis or neuralgia of the cardiac plexus. The existence of these two forms, which may sometimes be combined and

which may not always be easily distinguished, has led to great confusion as to the real nature of the true disease.

Angina pectoris is not a common disease. Eichhorst quotes the statement of Gilbert Blanc who treated 3,835 patients during ten years in St. Thomas's Hospital, London, without seeing a single case of angina pectoris. But the disease is really not so rare as this, and the frequency of the occurrence is better represented in the statement of Fraenkel, who had two distinct cases of angina among 250 patients in the course of a year.

Gauthier, from his analysis, found that of 71 cases, but 3, that is, 4.2 per cent. were cases of true angina.

The disease is rare in childhood, though v. Dusch saw a case in a boy aged eleven, and Blache a case in a boy aged fourteen, associated in one instance with calcification of the coronary arteries, in the other with affection of the aortic valves.

Though intimations of angina can be found in the writings of Morgagni, Hoffmann, and Sénac, the credit of having distinguished and named the disease is due to Heberden. Although Heberden saw arteriosclerotic changes in the aorta in his post-mortem examinations, he did not recognize the connection between stenocardia and sclerosis of the coronary arteries. Heberden's chief merit consisted in separating the condition from the ordinary cardiac dyspnœa or asthma which occurs during the later stages of heart disease. Heberden, who thus first associated the main symptoms, considered the affection a spasm of the heart. This view found able advocates in Latham and Dusch, and in still later writers. Brunton goes so far as to liken the condition to the muscular spasm of a distended stomach or bladder in the attempt to overcome an obstacle at the orifice of exit. The obstacle to the efflux of blood under contraction of the heart may be immediate, at the aortic orifice, or may be distant, in a general arteriosclerosis. But this view is not always reconcilable with the condition of the pulse which may show, as will be seen, every form of irregularity or may be perfectly normal. Parry and Stokes attributed the paroxysm to insufficiency of the heart, and Balfour lays stress upon the fact that the attack occurs immediately after some demand beyond the capacity of the heart to supply. Trousseau located the disease in the nervous system, and considered the explosion, like an aura, to be a manifestation of epilepsy. Parrot, Liégeois, Charcot, and Le Clerc connected it with hysteria. Elsner, Schmidt, Hasse, Darwin, and Vigieus considered it a manifestation of gout or rheumatism. Traube tried to establish a more mechanical theory, ascribing the pain to a compression of the nerve elements in the muscular tissue from over-distention of the heart cavities. Eich-

wald interpreted the pain as an overstrain of the muscle in the attempt to overcome an obstacle. As certain cases, even fatal cases, have been found without discoverable anatomical lesion, Romberg discards all mechanical theories and considers angina pectoris as a neuralgia of the heart. Piorry and Cahen had already defined it as a brachio-thoracic neuralgia. In fact, angina was so definitely determined to be a neuralgia that the only question of dispute was as to the nerve affected. Desportes located it in the vagus, Laennec in the sympathetic, Jolly in the intercostal nerves, Bouillaud in the phrenic nerve, Kirsch in the spinal cord, Romberg in the cardiac plexus. v. Basche distinctly subscribes to this view with the declaration that the essential element is a neuralgia. He bases his opinion upon the fact that the pains and sensations irradiate like those of a neuralgia, and that pains of every severity may exist without demonstrable anatomical change. As the evidence of neuritis has been demonstrated in several cases in the cardiac plexus, more especially by Peter and Lancereaux, this plexus may be considered the seat of the neuralgia and the source of the angina pectoris. Liebermeister also considers angina as a neuralgia, the pain of which has a particular quality and a more ill-defined character, in that it is not so strictly localized and has an associate feeling of syncope which usually attends neuralgia of the internal organs. Peculiar sensations are common to affection or injury of internal organs such as the testicle, ovary, etc.

From the multiplicity of opinions thus expressed it is evident, as stated, that different conditions have been described under the term angina, and Landouzy goes so far as to say that angina should not be regarded as a morbid entity at all, any more than an attack of epilepsy should be considered as an independent disease, a "maladie autonome."

It will be remembered that pain alone, however severe it may be or however much it may irradiate, does not suffice to constitute angina pectoris. The first observers noticed an additional element of far greater gravity than pain, to wit, the intense anxiety, the unspeakable fear of impending death. It may be admitted that no pain is so severe as that of angina pectoris, but it must also be admitted that other pains of nearly equal severity are entirely unattended with this indescribable anxiety. Cases of angina are indeed recorded in which the anxiety completely overshadowed the pain, fatal cases of quick syncope, the so-called *angina sine dolore*, in which there was no complaint of pain at all; and angina in less degree is often seen in other disturbance of the heart, palpitation, arhythmia, etc. This sense of anxiety impressed the older writers to such extent

that they incorporated it in the name of the disease as its most distinctive feature. Angina is literally a strangulation. The intention was to convey the idea of a constriction or choking of the action of the heart. The imminence of such a danger would instantly rouse into intense activity every nerve cell in the body, and the appreciation of this danger, which is of course cerebral, is conveyed in the term angina. The location of anxiety is, therefore, not in the cardiac plexus but in the cortical substance of the brain, and the essence of true angina is the recognition by the cerebrum of the terrible imminence of the danger to life.

Etiology.

True angina being dependent on organic change, usually of sclerotic or atheromatous character, is therefore a disease of later life. According to Quain, 80 per cent. of cases occur after the fortieth year of life. Of Forbes' 84 cases, 72 were over fifty. This factor of age was emphasized in the view of Rougnon and Baumès, that angina depends upon ossification of the costal cartilages.

As the causes which produce the condition upon which the disease depends (alcohol, syphilis, gout, high living, and hard work) are more common in the male sex, angina pectoris is much more frequent in men than in women. Lussana states that ninety-seven per cent. of all the observed cases occurred in men. Forbes cites 84 cases, of which only 8 were women, and Lartigue 74 cases, of which only 7 were women. Balfour saw evidence of angina in 98 of 581 senile hearts; of these, 17, or only one-sixth, were women.

Heredity plays an undeniable rôle in the development of arteriosclerosis, and cases (premature senescence) have been recorded in which angina existed also in the ancestry. Hamilton, Macbride, and Grisolle bespeak the influence of heredity represented by a predisposition to arteriosclerosis, and Huchard speaks of an hereditary "aortisme."

Leflaire remarks upon the frequency of attack among members of the professions, preachers, physicians, writers, etc. It is commonly said, too, that angina occurs with much greater frequency in the well-to-do upper classes and most commonly in fat subjects. It is not likely in these statements that the line was distinctly drawn between the cases due to organic diseases and the pure neuroses.

To arrive at a proper understanding of the subject it is necessary to hold fast to the view that true angina depends upon an organic disease that threatens dissolution by arrest of the action of the heart. The etiology lies chiefly in the direction of nutrition of the heart itself, to wit, in interference with the blood supply to the walls of the

heart in obstruction of the coronary arteries. All observers agree as to the frequency of affection of the coronary arteries and of organic change of the heart in true angina pectoris.

Experimentation would seem to indicate an easy way of determining the exact influence of occlusion of the coronary arteries, but the results of experimentation are not so uniform as to be conclusive. It is impossible also to estimate properly subjective sensations in the lower animals. The first experiments were made by Erichson (1842). Panum twenty years later succeeded in blocking the coronary arteries in dogs by injecting into the innominate tallow and wax. Occlusion of the coronaries produced in this way had the effect of arresting motion in the left auricle after the lapse of five minutes, in the left ventricle in seventy-five minutes, in the right auricle in ninety minutes; the right auricle (*ultimum moriens*) continuing to beat a few minutes longer.

Von Bezold made his experiments with rabbits by clamping the coronaries with forceps. The effect upon the heart was shown in retardation of the contractions in ten to twenty seconds, in arhythmia in forty-five to one hundred and fifty seconds, and in a short time thereafter the heart ceased to beat. Samuelson also noted diminished frequency. The right ventricle and auricle continued to beat some time after the left side ceased, with the effect of distending the left auricle to such degree as to make it appear as a swollen, glistening bladder. This extreme distention of the left auricle Samuelson regards as the cause of the pain and anxiety of angina pectoris. Cohnheim and Schulthess-Rechberg experimented preferably upon dogs, in whom the course of the coronary artery is more superficial, and the distribution without anastomosis is similar to that of man. These authors observed that ligation of a large branch of the coronary artery produced no immediate influence upon the activity of the heart. By the end of the first minute, however, the action becomes arhythmic and bradycardia is pronounced. In the course of the second minute both ventricles cease to beat simultaneously, and this event follows regularly without reference to the particular artery which has been ligated. After arhythmia has set in the action of the heart can no longer be restored. The complete paralysis which thus occurs was shown not to be due to anæmia, that is, to defective oxygenation, or to the accumulation of carbonic acid, but evidently to the effect of a heart poison, a product of metabolism which is in a normal state carried off in the course of the blood current, but which accumulates under defective nutrition (Fraenkel).

Michaelis confirmed in the hearts of dogs the conclusions of Cohnheim as to the effect upon curarized rabbits and dogs of occlu-

sion of the coronary arteries. Ligation of the left coronary artery in the dog is followed in two minutes by final arrest of the heart's action. This arrest is not caused by loss of blood and cooling of the heart, for the heart of the rabbit could be brought to beat again after arrest of the action. Cohnheim never saw œdema of the lungs in consequence of the operation, but Michaelis observed beginning œdema of the lungs in the rabbit and advanced stages of it in the dog. It was remarkable what slight injury would bring about perfect occlusion of the coronary vessels, but it was only after the lapse of half an hour that the first irregularities were observed in the action of the heart. This delay is explained by the fact that blood escapes into the cavities of the heart through the foramina Thebesii and new blood is introduced through the coronary arteries.

Leyden, who made a special study of diseases of the coronary arteries in connection with angina, attributed the peculiar heart pains and precordial anxiety to necrotic processes in the heart muscle, the direct anatomical effect of occlusion of the coronary arteries, while Potain finds cause enough for all the symptoms in anæmia, the result of a checked but not entirely interrupted circulation. Fränkel quotes extensively from Charcot, who describes the disease as a "claudication intermittente par oblitération artérielle." In this case the closure of a chief artery of an extremity, for instance of the common iliac, by local thrombus produces no effect so long as the patient is quiet, but so soon as any bodily efforts are undertaken, for instance a long walk, severe pains set in in the leg, associated with coldness, a feeling of deadness, and spasmodic contraction, with the visible signs of motor weakness. Such a condition exists in the heart whose coronaries have been closed by a sclerotic process, whether at the orifice or in the course of a chief branch. An increase of activity from any cause, emotional excitement, physical strain, defective oxygenation, perhaps the temporary accumulation of toxins, precipitates the attack of stenocardia, with irradiation to sensitive nerve elements, that is, it produces the violent pain with anxiety and insufficiency.

The irradiation is explained by the nerve connections of the cardiac plexus, as of the anterior branches of the four upper cervical nerves and the first thoracic nerves; of the first thoracic nerves and the lower fibres of the brachial plexus, etc. Huchard tabulates the reflexions as follows: Irradiation occurs (1) preferably to the upper extremities, and especially to the left arm; (2) to the cervical plexus with pains in the neck, face, tongue (Trousseau), chin, and ear (Butter, Wichmann), to the temporo-maxillary articulation where it may produce a kind of trismus; (3) to the external cardiac branches

of the pneumogastric, to the throat, larynx, stomach, and liver, producing aphonia, a kind of globus hystericus, a sensation of heat in the epigastrium, nausea, eructation, vomiting, gaseous distention, pains in the hypochondrium simulating hepatic colic; (4) to the intercostal and diaphragmatic nerves, with pains in the thorax, vertebræ, breast, and hyperæsthesia in the mammary region, as observed by Laennec; (5) to the hypogastric region (Blackwell), to the testicle with swelling (Hoffmann, Laennec, Gintrac), ilio-scrotal neuralgia (Axenfeld), strangury (de Lorme), dysuria (Blackwell, Lartigue), and (6) to the lower extremities (Friedreich). Irradiation occurs chiefly to the left because, according to Eulenburg, the heart and aorta lie to the left, or perhaps, as Fraenkel urges, because the attacks emanate especially from the left half of the heart.

The anxiety is, as stated, the appreciation by the cerebrum of the imminence of the danger, and is shown in certain affections of the cerebrum (typhoid fever, delirium tremens, melancholia attonita, etc.), and in other diseases of the heart (endocarditis, valve lesions, degenerations), entirely independent of pain.

The sudden death is explained by the paralysis of the heart under the action of accumulated toxins.

Morbid Anatomy.

Edward Jenner, the discoverer of vaccination, was the first to suggest the fact that angina pectoris was caused by occlusion of the coronaries, in a letter which he addressed to Heberden in 1778, but which he did not send out of consideration for the feelings of his friend, John Hunter, whom he rightly believed at that time to be a sufferer from angina (Fagge). Jenner (1775) first saw the calcification of the coronary arteries in an autopsy which was being made by John Hunter. At a subsequent autopsy in a similar case he was so struck with the feeling of mineral matter in cutting the heart with the knife that he looked up to the ceiling to see if some of the plaster had not fallen upon the organ. Parry soon afterwards first remarked upon the coincidence of coronary sclerosis with angina pectoris. Virchow, Cohnheim, and Quain laid especial stress upon embolism of the coronary arteries. Potain in 45 observations saw contraction of the two coronaries 20 times. Gothair and Huchard observed in 70 autopsies change in the coronaries 38 times, change in the aorta 17 times, change in the myocardium 4 times (Petit). The grave and intense cases are especially liable to show affection of the coronary arteries. Thus Wilde found atheroma of the coronaries in every one of six cases which ended in sudden death. Osborne found in a fatal case rupture of a coronary artery with effusion of blood into the pericardium.

The objection that cases of true angina occur without any lesion of the coronary arteries is met by the statement that other heart lesions may have the same effect. This is especially true in connection with the aorta and its valves. Arteriosclerosis is the predominant lesion, so that the condition is found more especially in connection with insufficiency or stenosis of the aortic valve, with aneurysm of the aorta, especially the ascending portion. Corrigan first called attention to the affection of the aorta, which was so well appreciated by the time of Wunderlich as to be regarded as the real cause of the disease. Auscher reported a case of a man, aged fifty years, who had suffered from typical attacks of angina pectoris for eight years, but upon autopsy the coronary arteries were found absolutely normal. There was considerable hypertrophy of all the heart walls, stenosis of the mitral orifice, and calcification of the aortic valve. Atheromatous processes are frequently found in the aorta about the orifices of the coronary arteries, and in some cases fortuitous deposits have actually blocked the mouths of these vessels. An exquisite case of this kind was reported by Potain, who found in the body of a man who died of angina pectoris at the Hôtel Dieu only two small plaques of aortitis, exactly localized at the orifice of the two coronary arteries. Huchard depicts a case of occlusion of the coronaries under a more general atheroma of the aorta. Cases of partial occlusion of one vessel are not at all infrequent. Balfour recorded the case of a man, aged twenty-four, who died after four months' illness with paroxysmal pain, in whom was found, as had been diagnosticated during life, at the base of the aorta a ring of atheroma, which greatly narrowed the mouths of the two coronary arteries. In the majority of cases the sclerotic process begins in the ascending aorta at the point of origin of the left coronary artery, and the lumen of the artery is often so much lessened as not to admit the entrance of a bristle. Curschmann saw two cases in young individuals of sudden death from angina where the sclerosis was limited to the orifice of the coronary arteries, which was so contracted as scarcely to admit the end of a bristle. The course of the vessels was perfectly sound, and excepting a few atheromatous plaques at the origin, there was no alteration of the aorta.

In other cases, and this is the rule, the thickening and calcification affect only certain branches, especially the ascending branch of the left coronary artery, which runs to the anterior surface of the heart. In consequence of such a sclerotic disease, the circulation may be entirely closed by a thrombus or an embolus. "When a large section of the left ventricle is thus cut out, momentary arrest of the heart's action in diastole must take place. The literature shows a

number of such cases with accurate report of the autopsy, among which cases that of the distinguished sculptor, Thorwaldsen, has a certain celebrity. Similar cases were reported by Laveran, Hammer, Tantain, Dehio, and Samuelson. Leyden also gives examples. Finally Huber published the autopsies of a large number of cases from the Pathological Institute at Leipsic, where death occurred in the same way" (Fraenkel). Tacchi reports the following lesions observed in 70 cases: Lesions of the coronary arteries, alone or accom-

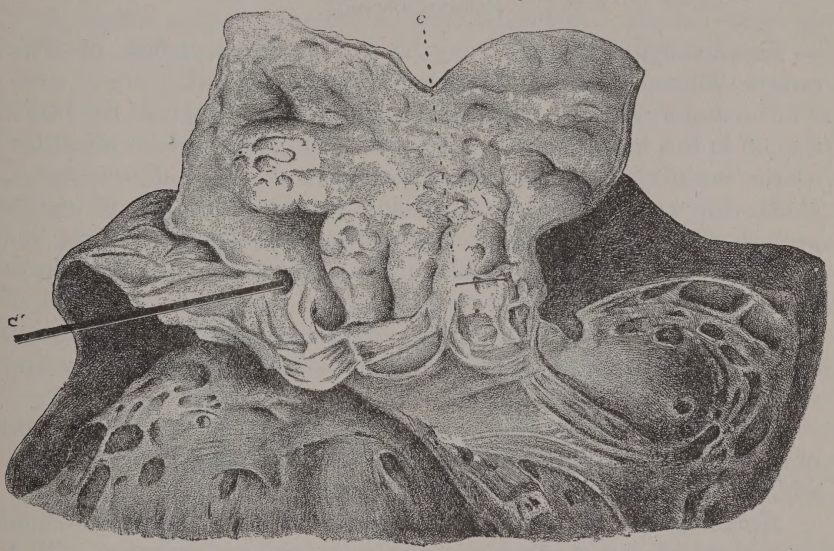

FIG. 29.—Angina Pectoris. *C* and *C'*, orifices of left and right coronary arteries. Sclerosis at orifice of left coronary artery. General atheroma of the aorta. The glass rod is introduced into the open right coronary artery. The obliterated left coronary barely permits the penetration of a bristle. The black dots below and within represent the orifices of supplementary coronaries. (Huchard.)

panied with other lesions of the heart or great vessels, 38 times; insufficiency of the aortic sigmoids, 12 times; aneurysm of the arch of the aorta, 5 times; fatty degeneration of the heart, 4 times; hypertrophy and dilatation, 4 times; pericarditis, 3 times; suppuration in the mediastinum, 1 time; negative results, 3 times.

So much then for the nature of the affection and the attempt to explain the symptoms by the character of the lesion.

At the same time it must be admitted that sclerosis of the coronary arteries may exist without angina pectoris. In some of these cases, at least, the sclerotic process did not entirely occlude the vessel. Every pathologist is familiar with the fact that arteriosclerosis of other vessels, radial, temporal, or cerebral, is not incompatible with blood supply sufficient for all ordinary needs, and even with long life

under favorable surroundings. A few other cases find explanation in the fact that the circulation is carried on through supplementary coronaries which remain unaffected. Such cases have been recorded by Tapret and Bador. Further, it is not disputed that severe, even fatal angina may exist in the entire absence of sclerosis of the coronary arteries. All the cases of so-called pseudo-angina are independent of organic change.

Pseudo-Angina.

Pseudo-angina pectoris may be due to direct, reflex, or toxic cause. These causes may operate alone or combined.

The *direct causes* are those which act upon the nerves of the heart, tumors in the neck or chest, aneurysm, enlarged gland, or affections of the brain or cord which implicate the origin of these nerves. Under direct causes may be included thus the paroxysms of angina pectoris which occur in the course of tabes dorsalis, constituting the so-called "cardiac crises" of Charcot. Leyden maintains that these attacks are caused by direct implication of the cardiac branches of the pneumogastric nerve, basing his opinion upon the not infrequent coincidence of gastric and cardiac crises, each depending upon changes in the pneumogastric nerve.

Hysteria and neurasthenia may be considered as the direct causes of the simpler form of cardiac neuralgia with more or less dyspnœa, which is sometimes distinguished as a pseudo-angina.

Reflex causes are those which result from disease of distant organs, as of the stomach or intestine, angina dyspeptica. Mere gaseous distention of the stomach or of the colon may suffice in nervous subjects, by mechanical interference with the action of the heart, to produce precordial pain with disturbance of respiration and extreme distress with anxiety, sometimes amounting to collapse and syncope. The "heart burn" of dyspepsia expresses the frequency of the association in the popular mind. So, too, diseases of the uterus and ovaries are sometimes associated with heart pains. Landois speaks of the "angina pectoris reflectoria" from irritation of the sympathetic fibres in the abdomen. Bergson relates a case where anginal attacks ceased under treatment of an enlarged liver. In noticing these cases Fothergill remarks upon the record in older literature of diseases of the abdominal organs being accompanied by attacks of angina. This author is so much assured of the connection that he will, he says, in all future cases, carefully look for sources of irritation in other viscera, and especially the pelvic organs. Brera went so far as to locate the disease in the abdomen and attribute it to pressure on the liver.

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NEWCASTLE ON TYNE.

The *toxic causes* are the most numerous. Chief among them is alcohol, which is the most fruitful cause of the changes which occur outside the heart as well as in the heart itself. Alcohol may, therefore, operate in every way, direct, reflex, and toxic, in the causation of angina pectoris. It is especially strong drink, brandy, whiskey, rum, or certain forms such as absinthe, which are liable to be followed by angina pectoris. The lighter wines, beer in whatever quantity, almost never cause the disease. Alcohol, next to old age, is the principal factor in the production of arteriosclerosis. Alcohol overstimulates and exhausts the heart. It impairs the power of the nerve centres, and finally raises the blood pressure and thus throws extra work upon the weakening or exhausted heart. Next in frequency ranks tobacco. A distinct form is sometimes set apart as the tobacco angina. The heart is extremely sensitive to the influence of nicotine. Retardation and irregularity of the pulse under the influence of nicotine has been noticed already in the discussion of other neuroses. Chronic nicotism shows, with various symptoms on the part of the nervous system (vertigo, insomnia, agoraphobia, loss of memory), the peculiar depression which constitutes a characteristic psychosis, known as the nicotismus mentalis, a tendency to impotence, palpitation, arhythmia, and angina pectoris (Dornblüth). Besides its effect upon the heart, tobacco has a most extraordinary power to contract the vessels, and perhaps there is no other drug that in a somewhat large dose can raise the blood pressure so rapidly and so much as nicotine (Brunton).

It is not so much the smoking too much as the smoking of very strong tobacco, which produces the attack, especially the smoking of imported pure Havana cigars, and the neuralgia is much more apt to occur when the cigar is consumed up to the smallest stump, and still more if the cold stump saturated with saliva is held in the mouth and some of the juice is swallowed (Pfungen). Exquisite cases of angina from the use of tobacco, where the etiology was established by the cessation of the attack on the abandonment of the use of tobacco, are cited by Beau, Championnière, and Blatin. Eulenburg mentions the occurrence of angina in a young cigarmaker who had smoked a large number of strong cigars every day for several years. The writer of this article was compelled to abandon the use of tobacco absolutely on account of nocturnal attacks of severe pseudo-angina five years ago.

The abuse of tea and coffee may lead to the same result. Professional men who resort to these stimulants to study or to secure hours of vigil, are finally compelled to abandon them on account of threatening angina. Stokes tells of a patient who was accustomed

to consume the night in literary and scientific work, and who took to help him every evening a large quantity of strong tea, and suffered in consequence of it violent attacks of precordial anxiety with a feeling of imminent death, which attacks ceased when the abuse was stopped. Coffee is itself a powerful stimulant to the heart, and the excessive use of it exhausts the heart in the long run. Under the head of toxic causes are to be mentioned finally the influence of gout, angina erratica, and of lead-poisoning, angina saturnica, both of which causes operate also, perhaps chiefly, in the production of arteriosclerosis.

Thomson recorded three cases of stenocardia after influenza. One of these cases was a man, aged thirty-nine, an excessive smoker. The disease terminated fatally in twenty hours. The symptoms on the part of the heart were those of a relaxed and fatty heart. The father of this man had died of angina pectoris. There was in no one of these three cases arteriosclerosis, enlargement of the heart, albuminuria, or diabetes.

Symptoms.

True angina sets in suddenly as a rule; that is, the pain and anxiety which distinguish the disease occur in a paroxysm or explosion. But other evidence of arteriosclerosis in the general failure of health and strength may have existed for years, or the heart may itself have shown signs of failing nutrition in general weakness, palpitation, dyspnœa, syncope. Gairdner declared that in not a few of the cases observed by, or more or less intimately known to, him, there was reason to believe that considerable suffering or sense of disability, though not always of one and the same character, had been present, and in some of these it might easily have passed unrecognized. In the majority of cases the patients are surprised by the attack, though it occurs more frequently in connection with or as a consequence of some bodily effort, as in breasting the wind, climbing hills or stairs, or after some emotional disturbance or exposure to cold. "Seldom at first can it be imputed to any obvious cause but undue bodily exertion. And for considerable period (perhaps for months, perhaps for years) seldom can it be imputed to anything besides this undue bodily exertion or sudden or strong agitation" (Latham). In one of Stokes' patients the slightest muscular effort, that simply of covering his feet in bed, sufficed to precipitate an attack. The patient therefore went to bed wearing his clothing. In the course of time the act of eating sufficed to produce an attack. The unfortunate patient was now compelled to eat his meals in his room, walking up and down. Dickinson reported the case of a soldier, aged thirty-five, who

was seized with and succumbed to an attack during the act of coition. More rarely, and usually later in the course of the disease, the attack occurs during perfect rest, even during sleep.

In a true angina the pain sets in suddenly and is usually indescribable in its intensity. The pain may, however, vary in every degree of severity. There are cases, as stated, in which syncope or death is so sudden as to leave no time for appreciation of pain, *angina sine dolore*. In other cases the pain is a dull aching sensation, which is overshadowed by the intense anxiety. In most cases the pain is excessive and agonizing, beyond the power of words to describe. It is associated also with a feeling of constriction, "as if a mailed hand grasped the chest in the cardiac area and squirted through its fingers flashes of excruciating agony up to the left shoulder joint" (Balfour). The features are pinched, the lips are blanched and set (facies Hippocratica), the forehead is covered with a cold sweat. Every movement is at once arrested, and the face shows the aspect of extreme anxiety, the anguish, which the patient is unable to express, associated with the appalling fear of impending death. The pain is felt in the region of the heart, but is rarely confined to this region alone. It irradiates as a rule to the left shoulder, frequently to the elbow, and sometimes along the ulnar nerve to the third and fourth fingers of the left hand. In rarer cases the pain irradiates also to the right shoulder, and may even follow the same course in the right arm. Still more exceptionally the pain irradiates backward to the scapulæ and to the back of the neck, implicating even the occipital nerves. Sometimes the pain shoots downward to the pelvis and even to the lower extremities.

Vaso-motor disturbances may attend the attack. The motor nerves are seldom affected, but vaso-motor disturbance is not unusual. The arms and fingers become pale and sometimes show a real syncope and asphyxia. Heberden reported such a case of asphyxia of the left forearm and hand, in which the extremity would become suddenly cold and insensible, with a sensation of swelling, during the stenocardia. Trousseau likewise reported a case where extreme pallor was followed by a pronounced violent and bluish coloration of the arm and hand; the natural color was restored as soon as the pain ceased. Von Basche said he knew of one case in which the patient said he felt as though cold water was being poured over the lower extremities; fraenkel speaks of cases in which the symptoms on the part of the heart are subordinate to sensations in the upper extremities, as of cold, numbness, deadness of the arm and skin, associated with pricking and creeping. Nothnagel, indeed, distinguished a special form as the vaso-motor angina, dependent upon

arterial spasm. In these cases the sensitiveness of the extremities is reduced, so that puncture with a needle or impressions of temperature are felt but indistinctly or not at all. The influence of cold is strongly felt in this condition, as even washing the hands in cold water may provoke an attack, and also coldness of the feet, low temperature of the room at night, a cold bed, may precipitate paroxyms. As these cases are entirely independent of arteriosclerosis, they fall under the category of the pseudo-anginas. Balfour records the case of a lady whose attacks were always preceded by pallor of the face and fingers. She could move about freely during the attack, and the suffering was always relieved by stimulants or by amyl nitrite inhalations. This was clearly a case of pseudo-angina of vaso-motor origin.

The breathing may not be at all affected, though it is generally somewhat hurried and superficial. Anything like real dyspnœa (cardiac asthma) indicates a complication on the part of the heart. With the superficial breathing that comes of the instinctive restraining of every effort, the patient is able at any time to take a long breath, but does not dare to make the experiment. In a case described by Hirschberg, the patient, a woman, aged forty-eight, when seized with an attack was compelled to sit up immediately in bed and bend the body forward. She breathed now rather more rapidly but without anything like dyspnœa, while a distinct cyanosis overspread the face and the visible mucosæ. The face was covered at the same time with a cold sweat.

The heart is not necessarily affected, though disease of the aortic valves is not infrequent; sometimes, as stated, atheroma of the aorta may be the whole cause of the affection. In such a case the heart would show the anomalies pertaining to affection of the aortic valves. Disease of the mitral valves cannot engender angina, and any evidence of affection of these valves would mean a functional disturbance (anæmia) or a mere coincidence. Nevertheless, as angina is an affection of advanced life, it is not surprising to learn that most cases show change in the heart. Walshe declared that he was able to discover physical change during life in every one of the twenty-four cases which he observed. During the attack the heart may be increased or decreased in frequency and force. Samuelson recorded a case in which the pulse sank to 30 beats in the minute. The old view, advocated by Heberden, made a strong action of the heart characteristic; the newer view, advocated by Parry, considers heart failure the most essential factor. But whatever the action of the heart, the blood pressure is always raised at the start. Brunton made repeated sphygmographic tracings of the radial pulse, finding increased tension of the arteries

during the attack. Eichwald noticed the increase in arterial tension in the pseudo-angina of severe hysteria, attacks of which are attended also with hardening of the arteries and the discharge of a large quantity of pale urine—urina spastica—an effect of high arterial tension. It is the rise in blood pressure which throws the extra work upon the heart and is the actual exciting cause of the attack. True angina distinguishes itself by elevation of pressure with normal frequency of the pulse, attacks occurring usually at a pressure of 170–230 mm. In these cases the rise of blood pressure need not be universal, as while the heart is struggling to overcome an obstacle registered by rise of pressure in the radial artery to 170–230, the pressure in the temporal artery may be no more than 120–140. During the interval between the attacks the pressure in the radials may be normal. This pressure in the adult, even up to advanced age, is 80–100 mm. Anything above this degree is suspicious (Federn). To estimate properly the blood pressure observation must be made during the attack itself.

During the attack the patient instinctively seeks the posture which will give the most relief. This relief is best secured in the upright or sitting posture. Huchard declares that every patient affected with angina prefers instinctively the upright posture. The recumbent posture, and sleep itself, augment arterial tension; the upright posture diminishes it.

The attack lasts from a few minutes to the greater part of an hour or more. The average duration is from five to ten minutes. In one of Huchard's fatal cases it lasted as long as thirty-six hours. These protracted cases are sometimes looked upon as successive attacks in quick series, or subintrant forms.

In some cases death is instantaneous. Walshe mentions the case of a patient who had been reading in bed. He was found dead with the thumb and forefinger in the pamphlet he had been reading, the bed clothes being also quite undisturbed. Fagge relates that the distinguished divine, Dr. Chalmers, retired to rest at the usual time on Sunday night after a day of labor, and the next morning was discovered dead and cold. The body had an attitude of calm repose, the bed clothes were scarcely disordered; on them rested a basin which had received the contents of the stomach. This was the only evidence of anything like a death struggle. The celebrated sculptor, Thorwaldsen, died suddenly in the theatre, and the diagnosis of angina was verified upon autopsy.

In order to prevent the annoyance of a coroner's jury, the writer was summoned, during the correction of the proof of this article, to establish death which had occurred to one of his patients, a man, aged sixty-four, who had been a sufferer from angina pectoris for

several years and who had fallen dead, apparently instantaneously, in the act of unrolling a bandage from his leg.

Sometimes the very first attack is fatal in this way. One of Huchard's patients was playing billiards. Suddenly he became pale, then livid, and fell dead without a word. Another stopped suddenly in the midst of a conversation, and fell dead without finishing his sentence. Sometimes death occurs in bed so rapidly and silently that an individual sleeping with the patient has neither seen nor felt anything out of the way. A number of the cases of sudden death from so-called heart failure, are really cases of angina. Dr. Arnold, of Rugby, was awakened from his sleep in his first attack, which proved fatal in the course of a few hours. In this case the heart was found large. The walls of the ventricles were thin and soft. A few slight atheromatous deposits were observed in the descending aorta. There was but one coronary artery, and considering the size of the heart it appeared to be of small dimensions. It admitted with some difficulty a small director.

As a rule, however, the patient recovers from the first to succumb in some subsequent attack. Sometimes the victim falls with the cry, "Oh, my heart!" Sometimes there is not even time for a cry, the patient falls in the act of placing the hand upon the heart. But as a rule the patient recovers from the first to succumb in some future attack. The intervals between these attacks are very varied. The case of the eminent surgeon, John Hunter, as narrated by Fothergill, is a striking illustration. He had his first attack of angina in his forty-fifth year; three years later he had the second attack; he had no third attack until 1785, when he became subject to paroxysmal attacks, which assumed more and more the character of typical angina pectoris. After suffering dreadful agony he would fall into a syncope which would last ten minutes, and from which he would awaken without the least recollection of what had happened. He became deeply sensible of the risk to which he was exposed by over-exertion, and still more by his uncontrollable temper, and was accustomed to say that "his life was in the hands of any rascal who chose to insult him." It was not long before such an incident occurred. He was flatly contradicted at a board meeting in the hospital, left the room in a rage, had just gained the next room, when he gave a deep groan and fell down dead. The heart was found small. The coronary arteries were converted into open bony tubes. The carotid and vertebral arteries in the cranium were also bony, and the basilar artery had opaque, white spots very generally along its coats.

Sometimes the patient is able to move about. Fraentzel speaks

of the case of a man, aged seventy, who was attacked in a store, out of which he went and leaned himself against the wall of a house until the intensity of the attack passed over. He then walked slowly with a facies Hippocratica, appreciated by every one who saw him, some distance to his house.

The attack may cease as suddenly as it began, leaving usually languor and prostration, and a feeling of numbness or formication in the arms. The attacks of pseudo-angina are liable to be attended and followed by eructations of gas and discharge of large quantities of urine. The first attacks are usually far apart, later attacks become more frequent. The first attacks occur only in the day, later attacks occur also at night.

Diagnosis.

The diagnosis of a marked case is easy. The agonizing pain, the unspeakable anxiety occurring in paroxysms and in an individual usually over fifty, who has, as a rule, shown signs of failing action of the heart, or other evidence of arteriosclerosis, make an association of symptoms which distinguishes the disease. The question of utmost importance is the recognition of organic disease and the separation of cases of purely nervous origin. To make this diagnosis Huchard tabulates the following aphorisms:

(1) Every angina produced by an effort of any kind, by rapid walking, etc., is a true angina.

(2) Every angina which occurs spontaneously, without the intervention of an effort, is a false angina.

(3) But an angina which occurs in the night, though independent of effort, is a true angina.

Hysterical angina shows itself most frequently in the female sex at the age of, and more exclusively as the result of, emotional disturbances. Attacks occur in connection with or as a consequence of anæmia, after parturition, lactation, abortion. The angina may be as sudden and as severe, but it is not so strictly localized. It does not confine itself to the heart or to the left arm, particularly to the region of the ulnar nerve, but may involve the whole side of the body. Not infrequently the pain begins in other parts of the body. Any association with hemianæsthesia, hysterogenous regions or zones, clavus, or other distinctive signs, would form even more characteristic features. The pains are also more liable to be attended with palpitation.

The emotional features continue throughout the attack. In hysterical angina the patient may walk up and down the room, throw herself on the bed or sofa, wring her hands or show manifestations of

chorea major. True angina permits no such manœuvres, for in true angina the pain and anxiety are so great as to inhibit every movement. Delusions and disturbance of special sense are more characteristic of pseudo-angina. Certain patients have a feeling as if the ceiling was about to crush them. They believe themselves doomed, that they must perish in half an hour or an hour. The delusion of the special senses takes the form that is sometimes seen in migraine. Thus there may be scotoma, phosphema, a turning wheel encircled by flames, or the chairs or tables or other objects of furniture may appear of gigantic size. These patients move in a state of constant apprehension and fear. The sight of workingmen or poor people on the street excites the fear of social revolution with murder and massacre. The crossing of a sash in the window pane across the street is looked upon as a cross over a grave. A strong man is regarded as an executioner, etc. These hysterical symptoms distinguish themselves from true angina by the absence of any constant and uniform blood pressure (Pfungen).

Neurasthenic cases occur likewise in younger subjects with other evidence of neurasthenia, excitable pulse, disinclination to effort, agoraphobia, or other anxiety, etc.; in the absence likewise of any evidence of organic disease.

The pseudo-anginas of alcoholism and nicotinism occur for the most part in younger individuals, and in connection with the history of excessive use of alcohol and tobacco, other evidence of which may be found in anomalies of the heart's action, in the mental and visual disturbance of tobacco, and in the gastric and psychical disturbance of alcohol.

The vaso-motor cases occur more especially after exposure to cold, and are distinguished by the prevalence of paræsthesia rather than of pain and anxiety. It is not so much the pain which predominates, as the spasm of the vessels in the extremities, with a feeling of coldness, stiffness, formication, anæsthesia, especially of the fingers.

As angina pectoris depends upon organic disease of the heart, a common source of confusion is with cardiac asthma. But dyspnœa is the cardinal symptom of cardiac asthma, while anything like a real dyspnœa does not at all belong to stenocardia. The dyspnœa of cardiac asthma is due to mechanical obstruction, which is expressed in other signs, as in cyanosis of the face, stasis in the extremities, swelling of the liver, sometimes by the expectoration of sputum stained with blood. The anxiety of cardiac asthma is more the distress associated with extreme dyspnœa, and the pain, which is such an agonizing element of stenocardia, is, if felt at all, only a subordi-

nate feature of cardiac asthma. Attacks of cardiac asthma are expressions of insufficiency which develop, like the paroxysms of angina, under increase of blood pressure with failing action of the heart. The heart failure in cardiac asthma is a chronic, often fatty, degeneration of the myocardium, attended by dyspnœa upon every effort, and by stasis, especially in the lungs. The paroxysm of cardiac asthma, which may be brusque in its origin, is a temporary collapse of the heart muscle, which cannot supply the additional sudden demand, and the collapse is shown in the rapid development of pulmonary œdema, as expressed in the extreme dyspnœa and in the moist râles which can be heard everywhere over the chest. These conditions are never seen in a case of pure angina. But combinations of angina with cardiac asthma, which are sometimes seen, may make the diagnosis very difficult.

Intercostal neuralgia, with its painful points, and pleurodynia with its increase of pain on inspiration, both affections prevailing with the entire absence of anguish, could not long be mistaken for angina pectoris.

Prognosis.

The prognosis is always extremely grave. The true disease has an anatomical foundation which is irremediable. Many cases, as stated, succumb in the first attacks, and every patient is in constant danger. At the same time it should be made plain to a patient that under proper management the severity of the attacks may be mitigated, and the interval may be increased, so that life may be prolonged for years. Individual attacks may be years, even many years, apart. In one of the cases reported by Murrell, the patient lived forty years after the first attack. Flint has known actual recovery to occur; that is, the circulation in the heart may adjust itself in some way. Curschmann described two cases in which the gravest possible attacks, which occurred with great frequency between the age of forty and fifty, disappeared entirely in the course of time, and left only a retardation of the pulse as a relic of the angina pectoris.

Treatment.

The treatment resolves itself into the treatment of the attack and the treatment of the interval. As the attack is announced by the elevation of pressure of the blood, which necessarily throws extra work upon the heart, the evident indication is to reduce this pressure as quickly as possible. We are more fortunate than our forefathers in the possession of remedies in the nitrites, which quickly

produce this desired effect. The most powerful of these agents is the amyl nitrite, which was discovered by Balard in 1844, and was first used by Guthrie in 1859. The inhalation of a few drops reduces the blood pressure at once by general relaxation of the arterioles, especially of those in the upper parts of the body. The dilatation of the surface vessels is indicated in the flushing of the face, which rapidly extends to the neck, while upon the breast appear red spots which rapidly increase and coalesce. At the same time the heart is set in active motion, and rapid pulsation is felt in the arteries. The remedy is best taken when the patient is in the upright posture, and is carried about ready for immediate use in the form of small glass tubes, devised by Solger, which may be crushed with the fingers so that the contents may be received upon a handkerchief and thus inhaled. The inhalation produces a sense of fulness and heaviness in the head, with a kind of intoxication without loss of consciousness. This effect is, however, very transitory. Longer inhalation develops vertigo with headache and stupor. The dose should be small at first, two or three drops, though it may be increased if necessary to as much as ten drops. The inhalation should be in all cases discontinued at once upon relief of the symptoms, and should not be pushed too long in case of failure. Unfortunately the remedy sometimes fails. Fagge reported a case in which the amyl nitrite was used without relief. See also myocarditis.

Should the amyl nitrite fail, resort may be had at once to ether and chloroform, though the older authorities were very timid in the use of anæsthetics. Ether, sulphuric or acetic, is to be preferred to chloroform, and it is best administered after the suggestion of Romberg, by pouring a teaspoonful or two in a saucer and having the patient inhale the fumes. Fraenkel knew an old physician who was seized every evening when he went to bed, and who learned to control the attacks by the inhalation of ether. It is now known that the deaths which occurred under the use of chloroform or ether should rather be attributed to the disease itself. But preference is in all cases to be given to the amyl nitrite, not only because it is less dangerous, but because the relief it affords is more immediate.

The other nitrites, especially nitroglycerin, are given more in prevention of attacks. Nitroglycerin is really an acid ether of alcohol glycerin with nitric acid, and as it liberates nitric acid *in statu nascendi* its effect is intense and it must be used cautiously at first. It produces the same result as the inhalation of amyl nitrite, but in a little longer time, as the effect is shown only after from three to five minutes. It has the advantage, however, that it continues much longer, sometimes as long as four to six or eight hours. It may be given first

in the milder cases of angina, as in a vaso-motor angina. It has the same effect also in dilating the capillaries, flushing the face, and producing fulness and heaviness in the head. But tolerance is soon established. It is conveniently kept in the shops in a one-per-cent. alcoholic solution, and the dose of this solution ranges from one or two to ten drops, beginning with the smaller amounts. The remedy may be also used subcutaneously in the same dose, with much more prompt effect. During the period of prevalence of angina, the remedy may be carried about in chocolate tablets, as devised by Rossbach, of which one contains 0.305 milligramme nitroglycerin. Of these tablets one to two may be taken daily. Murrell advises a dose every three hours regularly, with additional doses during the paroxysm. In one case Murrell pushed the dose up to 110 minims of the solution, that is, to more than one minim of pure nitroglycerin. v. Ziemssen speaks of an old major who formerly suffered such severe attacks as to prostrate him upon the street and who secured complete exemption for many months by the use of nitroglycerin. Hey found in the sodium nitrite a preparation of similar composition with amyl nitrite, and liberating, like nitroglycerin, nitric acid, which is really the essential agent. This remedy has, however, no advantage over nitroglycerin and amyl nitrite.

More protracted cases may call for the use of morphine, subcutaneously at first, and later possibly internally. All the best authorities on heart diseases warn against the use or abuse of morphine. Curschmann emphasizes the danger of strong narcotics during the attacks. He saw a number of cases of sudden death in angina which he was compelled to attribute to excessive doses of morphine. Electricity has been found of little value, but the application of the continuous current along the course of the vagus in the neck and down the arm in cases where a distinctly painful feeling is experienced in the hand, has been found useful in warding off attacks (Yeo).

Mild cases of pseudo-angina may be relieved by the antispasmodics, valerian, asafœtida, Hoffmann's anodyne, which lightly stimulate the heart. Counter-irritants to the surface, sinapisms, are of decided value. Fraenkel found in some cases relief from the quick application of cold in the form of ice-bags, or cold cloths in the region of the heart. Romberg once saw an attack cut short by the eating of ice cream. Schott applies heat in the form of hot water, 60°–70° C. (140°–157° F.), by means of a rubber bag constructed for the purpose, which is applied directly to the region of the heart. Sometimes this simple method furnishes excellent results.

The treatment of the interval resolves itself into the treatment of arteriosclerosis, especially by the iodides, and the administration of

tonics, such as strychnine and arsenic. Balfour declares that arsenic is particularly indicated in all heart diseases attended with pain.

Patients learn to prevent attacks by avoiding every exposure, indiscretion, and effort as much as possible. Under this head is included the learning of the difficult lesson of self-control.

These points in treatment are summarized in the response to a letter by Fothergill, quoted by Dr. Madden, who after mentioning the effect of amyl nitrite in reducing the duration of the attacks from twenty minutes to two in his own person, stated that he was finally able to discontinue all medication; but, he said, "On one point I have to be particularly careful, I dare not hurry myself in anything. If I do through inadvertence, I am speedily reminded of my mistake by an attack of pain."

DISEASES OF THE BLOOD-VESSELS.

BY

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LONDON.

DISEASES OF THE BLOOD-VESSELS.

DISEASES OF THE AORTA.

Inflammation.

THE walls of the aorta may be invaded by pus-forming micro-organisms and abscesses may result. Such abscesses, causing elevations of the internal coat of the vessel, have been described by Andral, Virchow, Spiegler, Schützenberger, Lebert, Rokitansky, and others. In all cases there were signs of septicæmia or purulent infection. The abscesses are analogous to those which under similar circumstances are sometimes observed in the muscular structure of the heart. Cases of suppurative inflammation of the aorta are very rare. It is seldom that the disease can be recognized during life, but it is noteworthy that in the case of a patient who manifests the rigors, sweats, rapid changes from very high to very low temperatures, and, perhaps, manifestations of purulent transformations in other organs, the tunics of the aorta may be attacked, and the aortic valves may be rendered incompetent.

The aortic endothelium also may be involved in ulcerative endocarditis. The ulceration may attack the aortic valves, already diseased, and spread up the vessel. Deep excavations are seen in some cases in the coats of the aorta. Ulcerative endocarditis may be brought about by several varieties of micro-organisms. In cases which are associated with pneumonia and in which the pneumococcus is the infective agent the aortic valves are especially liable to be attacked. Ulcerative disease due to micro-organisms may attack portions of the aorta already affected by chronic degenerations. In a case under my own care, of a woman, aged 62, who died with signs of heart failure and interstitial nephritis, there were several large ulcers of irregular form, with sharply cut edges, and portions of detached endarterium adhering at some points in the arch of the aorta and the descending portion, one ulcer being visible in the abdominal aorta just below the diaphragm. Sections from one of the ulcers showed clusters of micrococci in the deepest layer of the intima in the floor and edges of the ulcers. There were also chronic

changes in the aorta with calcareous degenerations. In some cases the two conditions have been found together—an ulceration of the valves or of the lining membrane of the aorta, and abscesses or collections of pus underneath the internal coat of the vessel. Ulcerative endocarditis, with exuberant vegetations on the aortic valves, may be accompanied by acute non-purulent inflammation of the aorta, as in a case under the care of Huchard in which, in addition to the ulcerative disease of the aortic cusps, due to pneumococci, there were patches of yellowish-green color, uniting to form a circlet above the valves, presenting the characters of acute aortitis described hereafter.

Acute and Subacute Aortitis.

Morbid Anatomy.—The aorta observed on its internal surface may present pale or bluish-white patches varying in size from a pin's head to a circle an inch in diameter, these often coalescing. The patches may be transparent, of grayish color, or opalescent and yellowish-gray and sometimes blood-stained. The finger passed over them finds slight elevations alternating with depressions. The elevated portions are soft and elastic. These may be isolated and with depressed borders, something like the swollen Peyer's patches in typhoid fever.

The appearance of dark blood-stains in the living membrane of the aorta must not be mistaken for inflammatory changes. These are due to post-mortem imbibition of the coloring matters of the blood. In inflammation the internal surface is paler than normal.

The patches may be gray and soft in the acute stage; yellowish and firmer in the later stages. The swelling is due to fluid exudation containing round or oval cells, which lift the inner coat and extend among the elastic fibres of the middle coat. There are evidently successive formations so that some present signs of recent changes, others show later transformations. The earlier patches have been termed "plaques gélatiniformes." The site of these appearances is chiefly the ascending and the transverse aorta; the disease process may be limited to this region, leaving the aortic valves intact. The lesions may be found also at those parts of the aorta whence the great trunks (the innominate or the left carotid and subclavian) emerge, surrounding these vessels with a sort of collar and leading up to an obstruction, more or less, of their lumen. (See Fig. 30, p. 465.) There may be narrowing of the coronary arteries; in fact the changes may be confined to their neighborhood, the adjoining endarterium showing small patches of inflammatory change encroaching on their lumen; there may be thus merely a peri-coronary aortitis.

The middle coat is rendered less resistant, so that it may distend with the blood-wave, though thickened by rounded or flat cells among its fibres. The aorta may dilate from deficient resistance of its walls, the dilatation being proportionate to the duration of the disease.

The external coat is also thickened and its blood-vessels are enlarged; numberless round cells infiltrate the fibrous tissue. Some vessels may rupture, causing visible hemorrhages. Sometimes the neighboring pericardium shows signs of dry pericarditis. Pericarditis with effusion has also been observed, though very rarely. There may also be peri-aortitis. The most important structures thus involved are the nerve-filaments or the ganglia which surround the aorta. Lancereaux noted in 1891 a case in which, after death in a paroxysm of angina pectoris, there were found inflammatory changes of the aorta in the internal coat constricting the coronary arteries, and there was an infiltration of inflammatory products in the external coat also which involved the nerve-elements of the cardiac plexus. He described a second case in 1894 in which, with aortitis in patches (*aortite en plaques*) which he considered due to malaria, there were signs of chronic aortitis, constriction of the coronary arteries, and manifest lesions of the nerve fibres of the cardiac plexus which were involved in the thickened external coat of the vessel.

Symptoms and Course.—Difficulty of breathing becomes manifested, with a sense of weight or constriction in the chest and distress or anxiety. The character of the dyspnœa is peculiar, inspiration being long and painful and expiration short. The recumbent position is not tolerated; there is some orthopnœa. The difficulties are increased by effort, but sometimes there are attacks of dyspnœa of moderate severity not provoked by exertion. There may be a dry cough. *Pain* is severe; at first it is a sense of constriction about the midsternum. Sometimes there is the feeling of a lump in the throat, the "globus hystericus." The pain in some cases is agonizing; there may be radiations to the neck or down the left arm, and the attacks of intense pain may have all the characters of true angina pectoris. It has been described in some cases as of a burning or tearing character behind the sternum. *Insomnia* is in many cases a distressing symptom. *Vertigo* has been noted in occasional instances. *Dyspepsia* is a symptom in almost all cases, there being frequent manifestations of flatulence with nausea and eructations, sometimes vomiting. The face is pale, or of the color of yellowish clay, the pallor increasing as the disease advances, or it may present a leaden hue; it wears the look of intense anxiety. In some cases forcible pulsations of the arteries of the neck have been noted. Usually the extremities are cold; slight pitting may be observed over

the tibiæ and in a minority of cases there has been advancing œdema. The disease is not attended by elevation of temperature.

A sign relied upon by Potain is an abnormal upward displacement of the subclavian arteries so that these are felt to beat in the hollow above the clavicle. The sign is most marked in the case of the right subclavian. This is due to the dilatation of the aorta, causing its branches to be lifted to a higher plane. In the normal state the subclavian artery does not rise above the clavicle; it may be two or three centimetres above the edge of the bone when the aorta has become dilated. Confirmatory signs should be sought for, viz., the enlarged outline of the vessel as shown by plessimetric percussion and the increasing loudness of the aortic second sound. Implication of the orifice by passive dilatation or by the extension of disease to the semilunar valves of the vessel will be evidenced on auscultation by the occurrence of diastolic or of systolic and diastolic murmurs in the aortic area.

Causes and Modes of Development.—Acute or subacute inflammation of the aorta may arise and progress as an independent disease or may occur as a dangerous or fatal engraft upon pre-existing chronic disease. It is clear that there may be repeated manifestations of the inflammatory processes; so the post-mortem appearances vary with the time which elapses before the fatal issue.

It seems from the somewhat limited evidence available that the protopathic lesion may be the result of some forms of infective disease in their late stages, sometimes long after the original infection. Small-pox has been cited as a cause by Brouardel, scarlet fever and measles by Huchard, typhoid fever by Landouzy and Potain. From my own experience influenza may be a cause. In a lady, aged sixty-five, who came under my care there had been no morbid antecedent until an attack of influenza which was followed by extreme nervous depression. Two years after the attack there was probably re-infection. There occurred intense sternal pain aggravated by even slight exertion and accompanied by nausea and sometimes vomiting, with dyspnœa. There were many other signs of dyspepsia, flatulence, pyrosis, constipation, and disinclination for food. The site of maximum pain was the third intercostal space to the right of the sternum; on attempted walking there were radiations through the shoulders. The pulse was very slightly quickened; the normal was 80, it was now 92 but perfectly regular, and there were no physical signs of disease of heart or arteries. There was no improvement under treatment and one month after my observation great agony at the sternum was experienced for twenty-four hours. The patient then slept, but woke during the night and became suddenly convulsed (as

her husband reported it) about the chest and neck for a few seconds only and then died. There was no post-mortem examination.

Lancereaux has cited malaria as a cause, but authorities of great weight (as Sir Joseph Fayrer and Laveran) have declared that they have never observed a case presenting good evidence of the association. I have seen two cases which confirm the view of Lancereaux. In the one, a lady of middle age, with no morbid antecedent except malaria from long residence in an aguish district, there were signs of aortitis with aortic incompetence. I have on many occasions noted the advent and progress of disease of the aortic valves in those who have lived in malarial districts but who have shown no other disposing causes to the disease. In a lady, aged 49, who had long resided in malarial districts in India and in Australia, but who herself had manifested no signs of ague, I observed signs of the onset and course of aortitis. There had been one attack of rheumatic fever fifteen years previously, and it is possible that some valvular imperfection was the legacy of this, but the course of the symptoms was that of acute aortitis, though perhaps engrafted on pre-existing disease. The symptoms began with dyspnœa on exertion and almost constant pain in the heart region, with occasional numbnesses in the left arm. The pulse was 100 per minute and very irregular. The outline of the heart and vessels was normal. A systolic murmur was heard near the sternum at the end of the second intercostal space and over the third cartilages, and up each carotid. In the course of eleven days the precordial dulness increased and exhaustion followed the slightest effort, even turning in bed. The diastolic murmur of aortic regurgitation was now manifested, though it was not present before. There were paroxysms of whistling breathing (inspiratory dyspnœa), then cough of a dry tickling character. The pulse-rate increased to 120 and over; dreadful pain was experienced across the chest and down the left arm. Œdema commenced and increased in the lower extremities and death ensued about four months from the first record of symptoms.

The other causes of acute cannot be differentiated from those of chronic aortitis. It would seem that, just as ulcerative endocarditis may become manifested in previously diseased endocardium, so the acute inflammatory changes of aortitis may be initiated by many toxic agencies in the coats of the aorta, internal and external, which have already undergone chronic morbid changes.

Diagnosis.—For the diagnosis of the disease in the early stages the most important consideration is the association of precordial pain and difficulty of breathing. Acute pain, constant or paroxysmal, over the site of the heart or aorta, though of extreme intensity, and hav-

ing radiations to the left arm, the back, and the shoulders, as in angina pectoris, is of no diagnostic import *per se*. I have recorded many examples of pseudo-angina with the accompaniments of palpitation (or tachycardia) or cardiac irregularity having nothing in common with disease of the aorta. It is probable that there may be even a neuritis of the cardiac plexus without involvement of the aorta. The pain of aortitis is of burning or tearing character, with a sensation of constriction or tension referred to the sternum. It may be almost constant or may be manifested only at intervals (generally lasting for some hours) or it may be accompanied by paroxysms having the characters of true angina pectoris with radiations to the shoulders or arms, to the back, or to the throat. The difficulty of breathing is essentially of the form of cardiac dyspnœa, dyspnœa of effort, though there may be paroxysmal attacks without provoking cause. The united effect of the distressing pain and the dyspnœa is intense anxiety which is imprinted upon the countenance. Corroborative signs are those which result from the obstruction to the blood-flow due to the narrowing of the vessels proceeding from the aorta. Impediment to the flow through the carotid is shown by the pallor or yellow-clay color of the face as seen in aortic valvular disease; so the face presents not only the characteristic look of anxiety but the hue of anæmia. Obstruction of the coronary arteries is evidenced by the increasing feebleness and tendency to syncope, by the manifestations of œdema, and by the other signs of heart-failure. The extension of the disease (in a more rapid manner than is observed under other conditions) is shown by the manifestation of systolic murmurs owing to obstruction at the aortic orifice, from involvement of the sigmoid valves in the disease or from roughening of the endarterium above the valves, or of diastolic murmurs indicating the advance of the morbid process inducing incompetence of the valves, or both these murmurs combined. Dilatation of the aorta may be evidenced by enlargement of the outline of the vessel as determined by plessimetric percussion. Consecutive dilatation or hypertrophy of the left ventricle may be determined by the usual physical signs.

The second sound of the heart may assume a metallic or tympanitic character and this is of importance as telling of a dilatation of the vessel which may not otherwise be detected; but it is not invariably the case. If the site of the disease be such as to involve the sigmoid valves these may become swollen and the sound of their tension will be diminished. It is then muffled and not accentuated, and after the lapse of some time a diastolic murmur may be heard after the dull second sound.

In regard to differential diagnosis the most practically important

is probably that from *hysteria*. In the case of a lady, for example, who complains of severe pain at the heart when the physical signs indicate no notable abnormality, the conclusion might be too quickly arrived at that all the asserted suffering was subjective. The quickened pulse, the "lump in the throat," the general agitation, the obvious dyspnœa might be common to both maladies. The difficulty of *inspiration* would lead one to suspect aortitis, but the clew would be given chiefly by the constricting character of the pain, the effect of effort, even slight, in inducing the symptoms of distress (the hysteric patient would perform movements that the subject of the former malady would refrain from), the expression of countenance betraying more clayey pallor and more abiding distress and differing from the "facies hysterica."

Huchard speaks of a difficulty in the diagnosis from *exophthalmic goitre* and cites cases in point. It must be very rarely that such difficulty can arise. The rapid heart which is an early symptom of Graves' disease is not attended with the severe pains and distress of acute aortitis, and there is usually a fidgetiness with excess of movement in this disease, while the subject of aortitis manifests a desire for repose and especially refrains from movements of the upper extremities. The diagnosis of Graves' disease will be confirmed by the advent of swelling of the thyroid, the proptosis, the eyelid signs, and the muscular tremors; while that of aortitis may be shown by the signs of progressing disease of the aortic valves or of coronary obstruction and consequent cardiac failure.

There may be a possibility of difficulty in the diagnosis from *pericarditis*, as pericardial friction may be heard over the base of the heart in aortitis. In the majority of cases the doubt will be solved by the observation of concomitant signs of acute rheumatism in the subject of pericarditis. It is very rarely that in the early stages of pericarditis intense pain of the character of angina pectoris is manifested, but this happens in some cases, and Andral has cited two examples.

Treatment.—The first indication is rest in the semi-recumbent position. The pain and distress may be greatly relieved by the application at intervals of ice-bags over the aorta. The ice-bag should be suspended from a cradle so that no unnecessary weight presses upon the chest. As an alternative to the ice-bag, sedative liniments may be applied, but frictions must be very gentle. The following plan has given me good results in all forms of precordial pain. Take of liniment of belladonna, liniment of aconite, and oil of peppermint equal parts and mix intimately with a quantity equal to the whole of benzoated lard. A creamy ointment results which

may be gently rubbed into the painful area by a pad of cotton wool, or a layer smeared therewith may be applied and covered by oiled, or vaseline-smeared, lint. If the pain and distress are not sufficiently controlled the local hypodermatic injection of morphine hydrochlorate (gr. $\frac{1}{6}$ to $\frac{1}{3}$) should be employed.

For internal medication the iodides (of sodium, potassium, or ammonium) are of the greatest usefulness. The commencing dose should be five grains three times a day, but this may be increased to ten or even fifteen grains for each dose.

Especially at the times of dyspnœa, but also as an adjunct to regular treatment, inhalations of iodide of ethyl or of nitrite of amyl are valuable. Five minims of ethyl iodide may be sprinkled upon cotton wool, and inhaled three or four times a day, or the inhalation may be repeated every half-hour during a dyspnœic attack. Great and lasting relief is often thus brought about and any uncomfortable symptoms of fulness in the head are much less than in the case of inhalations of amyl nitrite. The latter inhaled in five-minim doses is more effectual, however, in certain cases.

For the treatment of the insomnia chloralamid in twenty to thirty grain doses is often effectual and is harmless. Sulphonal is not without its dangers. The bromides in sufficient doses are often too depressant. Morphine is best administered by hypodermatic injection and with all caution against inducing the morphine craving.

Digitalis, caffeine, strophanthus, and the cardiac tonics generally are harmful in this disease. An exception may be made when the left ventricle is obviously becoming weaker and especially if œdema is manifested. Then small doses (five to ten minims) of the tincture of digitalis may be administered every four hours in conjunction with $\frac{1}{100}$-grain doses of nitroglycerin.

Chronic Aortitis—Atheroma.

Morbid Anatomy.—In chronic inflammation with the associated degenerative changes, the aorta is observed to have lost the smooth, uniform, supple characters which it possesses in the normal state. The internal surface when the vessel is slit up may be seen to present whitish or yellowish patches, slightly raised from the surface, but the eye detects the glistening and the finger the moist smoothness of the unaltered lining membrane of the aorta which still covers them; or there may be observed depressions and roughnesses in these patches, the endothelium having disappeared. The vessel no longer presents its natural curve but is dilated and manifests a bulging above the aortic valves, its external coat over the depressed areas showing sub-

sidiary bulgings. Or the dilated vessel may show porcelain-white patches, as well as those which are yellowish, their appearance being, as Morgagni expressed it, as of melted white wax dropped on a cold surface to harden there; the unwary finger passed over the surface may be scratched or cut by the hard and sharp edges of the calcareous plates.

The sites of these changes are most frequently the ascending portion and the arch of the aorta; with less frequency the descending

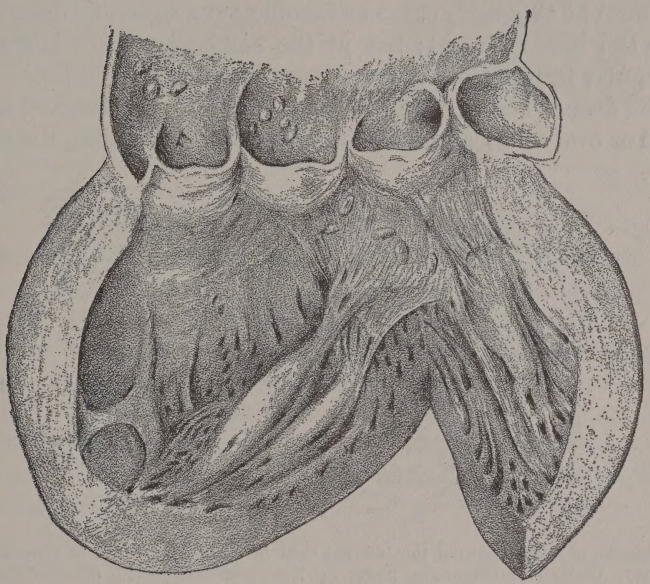

FIG. 30.—The Earlier Changes of Atheroma. Small elevations are seen on the large curtain of the mitral valve and on the lining membrane of the aorta. As a result of the concurrent changes in the fibrous tissues, the left posterior cusp is drawn upward, producing slight incompetency of the valve and partially occluding the orifice of the coronary artery. (Semi-diagrammatic.) (From a drawing by Miss Mabel Sansom.)

thoracic and the abdominal aorta. The coronary arteries of the heart are implicated in many cases.

Dr. W. Ainslie Hollis (*Journal of Pathology and Bacteriology*, 1894) finds that of all structures the sinuses of Valsalva are the most frequently attacked and the disease seems to start from the ridge bounding the upper edge of each. Upon the aortic semilunar valves the commencing spot is often just below a corpus Arantii, thence along a line bordering the lunule on either side, between the fibrous ridges which constitute a part of the normal structure of the cusp. When incipient atheroma has attacked the aorta the lesion frequently assumes the semblance of sinuous, elevated ridges of the endothe-

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lium, the pallid linear elevations interlacing and forming a delicate tracery just above the valve.

On microscopical examination in an early stage of the disease it is seen that the endothelium is unaltered in structure, though it is elevated by swollen tissue beneath it. It is, therefore, in the highest degree improbable that the disease starts from the lining membrane of the vessel and extends to the periphery. The fibres of the connective tissue of the deeper layers of the internal coat are opened out, showing abnormal spaces. The thickened wall of the vessel no doubt affords even at this early stage an insufficient support in these minute areas to the blood current; but at the same time there is a morbid and irregular hyperplasia of the connective tissue.

In the deeper layers of the internal coat may be observed clusters of round or oval cells; and these, with the fluid exudation, the opened-

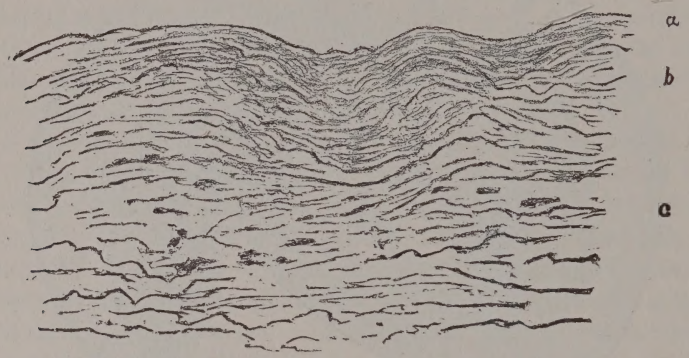

FIG. 31.—Sections of Aorta Showing the Internal Coat in the Earlier Stage of Chronic Aortitis (Atheroma). Under a low magnifying power. *a*, Normal endothelium; *b*, fibres of the internal coat separated, showing inclined meshes; *c*, degenerated patches and groups of exudation corpuscles in the deeper layers.

out meshes of the connective tissue, and the proliferating fibres of the latter are the causes of the swellings and elevations observed.

In the very early stage of atheroma, according to Dr. Hollis, the endothelium and basement membrane are invaded by many corpuscles which stain readily with basic dyes. With the advance of disease the corpuscular invasion extends centrifugally, "apparently following in the first instance the strands of connective tissue in their oblique passage among the fibro-elastic bundles of the inner and middle coats." These corpuscular swarms are, according to this author, "blood-born" cells; he terms them "nuclear bodies;" they are vagrant corpuscles from the blood. It is probable that the determination of such corpuscles to the affected spot of the lining membrane of the aorta is in consequence of the invasion of some foreign particles.

The nuclear bodies may be regarded as blood-scavengers which are observed when they have done their work; moreover they are probably originators of fibrous overgrowth. The corpuscular infiltration

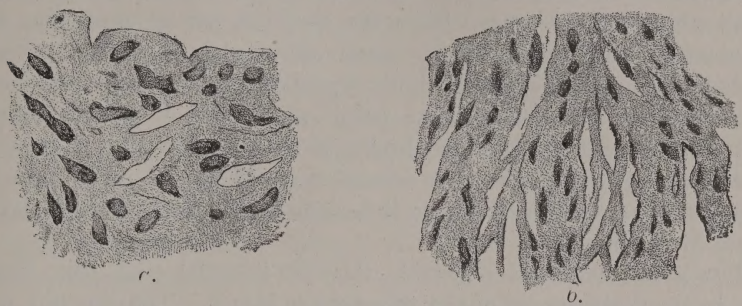

FIG. 32.—Sections of Atheromatous Tissues Showing Nuclear Bodies. In *a*, these seem to pass obliquely outward. In *b* is shown the dissociation of the fibrous elements of the tissue and the widening-out of the meshes following an invasion of the nuclear bodies. Highly magnified. (After W. Ainslie Hollis. Drawn by Miss Mabel Sansom.)

is followed by fatty degeneration. Fatty degeneration is never a primary affection, but is always preceded by, and associated with, the invasion of the affected portion by the nuclear bodies. In the case of an aortic semilunar valve attacked by atheroma, the normal fibrous bundles are dissociated; there is a widening out of the meshes following an invasion of the nuclear bodies.

Fatty degeneration commences very early in the deeper portions of the patch; on section an opaque yellow streak is seen in the layers bordering on the middle coat. The adventitious round cells as well as the neighboring normal cells break down in the fatty transformation, fine oil globules being first visible and in course of time crystals of cholesterin and margarin.

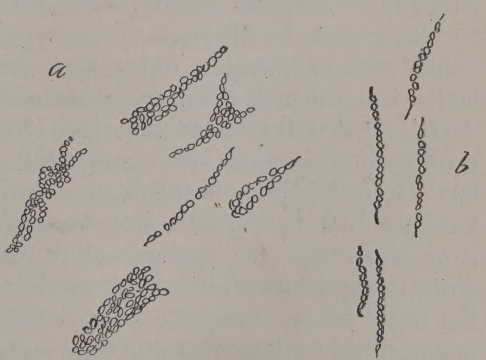

FIG. 33.—Fatty Degeneration in Atheroma; *a*, of the tissues of the internal coat; *b*, of muscle-cells from the middle coat. × 350. (Coats.)

The cause of these local infiltrations and fatty degenerations has been shown with great probability by Hippolyte Martin[1] to be an inflammation of the arteries of the arterial wall corresponding to the patches—the vasa vasorum—causing an obliteration of their lumen and consequent arrest of the nutrition of the tissues they supply, and degeneration of the cell elements in the affected area.

"If we examine attentively the minute arteries which penetrate the external coat of the vessel and course in all directions in the normal fibrous tissue we find them all healthy save those which correspond to the atheromatous area. Here the nutrient artery supplying the degenerated portion, especially when observed in a section perpendicular to its long axis, shows signs of proliferative endarteritis which, in the case of an atheromatous patch which has formed a cavity, obliterates almost entirely the lumen of the vessel so that it renders the circulation within the area almost impossible. This is a lesion which is never wanting but is often difficult to demonstrate" (Hippolyte Martin).

The arteries do not penetrate into the internal coat; this, therefore, in the early stages of the affection is intact. The morbid process is first manifested about the distal portions of the arterioles where circulation is less active, not in the external coat where are many collateral branches. Dr. Hollis agrees that the vasa vasorum are attacked at an early period of the disease, the tissues about the nutrient arterioles being especially beset with numerous deeply stained corpuscles.

It would appear to be the view of Dr. Hollis that the first step in the morbid process is the passage of some foreign particle or particles into a minute artery supplying a given area of the coats of the vessel; the next step in the process is the determination to the affected area of the nuclear bodies which act as destroyers of the offending particles. The view of Hippolyte Martin would rather be that the irritating particles first cause a more defined obliterative endarteritis of the minute branches of the vasa vasorum, and that the nuclear bodies are wandering leucocytes escaped from the diseased vessel.

It is generally agreed that fatty degeneration as well as fibrous proliferation are consequent upon the corpuscular infiltration. As the softened fatty material accumulates in the form of a cheesy or fluid product in the vacuoles formed by the broken-down tissue, it presses toward the surface and ultimately the endothelium breaks down to manifest an atheromatous ulcer, the débris being swept into the blood stream from the aorta. The appearance of the softened material has been termed atheroma—ἀθήρη, a porridge or gruel, corresponding with the "pulticula" of the Romans, the "bouillie" of the French, and the "pap" of the English. The patch may be infiltrated with lime salts, sometimes without, but usually in association with, the fatty change. The deposited calcareous granules become aggregated into hard plates. The aorta may thus be converted into a hard but brittle cylinder, the deposit of lime salts being chiefly in the middle coat. More frequently there is not this uniform deposit,

but hard plates are seen here and there presenting sharp borders which have pierced the internal coat. On these rough points or surfaces may form coagula of fibrin derived from the blood stream. The gelatiniform patches lately described as indicating acute aortitis may, in like manner with the more firm and fibrous nodules which are the usual commencing signs of the chronic degenerative process, undergo fatty degenerations and calcareous infiltrations; and as I have before said, the acute may be manifested in the subject of the chronic changes. In all cases in the later stages all the coats of the vessel are involved: the internal coat is infiltrated, degenerated, or eroded; the middle coat, though it escapes in great degree in the earlier stages, infiltrated, softened, and disintegrated; and the external coat dilated into one great pouch with many subsidiary ones, its fibrous tissue thickened, and its vessels dilated.

The coronary arteries may be obstructed by the disease in two modes—a patch of atheromatous swelling or of calcareous substance may encroach over their lumen, or by the changes in the fibrous tissue the aortic cusp may be drawn out of place in such manner as to obstruct the orifice of the vessel. In many cases the coronary arteries themselves in a part or in many parts of their course manifest patches of atheroma or calcareous change. In any post-mortem examination the coronary arteries should be opened up by scissors and their lining membrane carefully examined, for patches of coronary endarteritis are often overlooked.

Causes and Modes of Development.—Chronic aortitis with atheroma and calcification may be simply a senile change. In the post-mortem examination of any subject over sixty years of age it is most common for some signs of aortic atheroma to be found. In many there are extensive lesions, abundant calcareous incrustations in the vessel, and yet no morbid sign has been manifested during life to indicate their existence. Whether any such signs are manifested at all and what are their characters and significance are determined by the localities in which the changes take place and the portions of the vessel which they involve. If the degenerated and infiltrated patches are well above the valves, there need be no interference with the normal processes of life. If they are just above the valves and implicate these by extension, the signs of aortic valvular disease (obstruction or regurgitation, or the combined lesions) will be manifested. If the thickening and degeneration extend to the orifices of the coronary arteries, or if these are blocked by atheromatous patches in any part of their course, there will be the evidences of cardiac failure. The production of saccular aneurysms will be dealt with hereafter.

Another cause of chronic disease of the aorta is *muscular over-*

strain. The disease is met with more commonly in men than in women, and occurs especially in men whose occupations involve strong muscular efforts, continuous or intermittent. It has been shown to affect hammer-men, sawyers, smiths, bricklayers, hodmen, ship-porters, and soldiers accustomed to carry heavy accoutrements. In these it may be manifested at a comparatively early age. Clifford Allbutt[2] has shown that in a town where many of the inhabitants are accustomed to hard physical labor it often occurs in quite young men and it is as common a cause of organic disease of the aortic valves as rheumatism. It would be difficult to understand that excessive muscular strain could produce the lesions distributed throughout the various areas within the aorta if the affection, starting from a previously healthy vessel, were due to the direct effect of the increased pressure upon the lining membrane. It would seem that in a healthy aorta there would be an even distribution of pressure. The explanation is much more intelligible when we consider that the effect of the suddenly increased tension induced by the efforts is upon the terminal arterioles, distributed to the coats of the vessel. They are, as it were, between two pressures—the intra-aortic and the systemic—and the greatest strain would be manifested at their peripheral terminations in the capillaries distributed to the aortic wall.

A third cause of the disease is *gout;* this has been recognized by observers from very early times. Gout has been an antecedent in many cases of angina pectoris. Dr. W. T. Gairdner has said: "In gouty subjects the heart and arteries are very prone to become disorganized and the disorganization is specially apt to assume the form which other observations show to give a predisposition to angina, viz., calcareous degeneration of the aorta, especially at its commencement, and of the coronary arteries."[3] It is very probable that the form of aortitis induced by gout is particularly liable to affect the portion of the aorta just above the valves, and thus to encroach upon the orifices of the coronary arteries. It is rarely that one has the opportunity afforded by clinical investigation of tracing the effects of gouty storms in producing the lesions of chronic aortitis. I have observed the case of a gentleman, aged fifty-three, in whom symptoms of general enfeeblement, fluttering of the heart, and gradually increasing dyspnœa had been coming on for two years. He was the subject of gout and generally had three attacks a year. The early observations of his heart showed the systolic murmur at the base of aortic obstruction. The pulse was one of prolonged arterial tension. Six weeks after my first observation an attack of gout occurred and then a slight diastolic murmur became evident in addition to the systolic. This

diastolic murmur became more and more pronounced, the conditions of distress were aggravated, and the patient died about five months after his coming first under observation. It seemed clear that there had been gradual changes for many months in the aorta above the valves, implicating them only to produce some obstruction to the blood-flow. Then with the attack of gout there was a more rapid progress of the disease so that the valves became incompetent and regurgitation took place in increasing degrees.[4] In most cases the progress is so gradual that it cannot be traced; in many there are no outbreaks of acute gout and the development of disease of the aorta may be the chief or only sign. Lancereaux has recorded an interesting case of a woman, aged sixty-one, in whom after death there were found signs of interstitial nephritis (gouty kidney), acute and chronic aortitis with calcareous change causing a narrowing of the calibre of the great arteries proceeding from the vessel, as well as of one of the coronary arteries. The mitral valve was thickened and presented groups of vegetations dotted with grayish granules which were demonstrated by chemical and microscopical examination to be composed of uric acid. There seems to be a considerable probability that the endarteritis of the nutrient arteries of the aorta giving rise to the inflammations and degenerations of the vessel in gouty subjects is due to the determination of insoluble urates to their terminal branches.

Other causes to which chronic inflammations of the aorta have been ascribed are *over-indulgence in alcohol, excess in eating as well as drinking, and in smoking and chewing tobacco.* Most of these must produce their effects by causing a faulty nutrition and morbid tissue metamorphosis whereby irritant toxins are evolved. No doubt the effect of excess of tobacco-smoking or tobacco-chewing is to constrict the arteries, to induce abnormal intra-arterial tension, and to give rise to symptoms which resemble angina pectoris. It is not proved, however, that these habits alone have sufficed to induce acute or chronic inflammations of the aorta. *Lead-poisoning* is probably a cause though not a frequent one. Leudet has recorded one case of atheromatous disease and dilatation of the aorta out of twenty-four deaths after poisoning from lead.[5] Oliver,[6] of Newcastle, however, though he has noted the increased arterial tension which attends certain phases of chronic lead-poisoning, has not—and his experience has been very large—recorded any case of aortic diseases as resulting.

Syphilis is a potent factor in the production of chronic disease of the aorta. Its influence is often subtle, for it is manifested in the tertiary period very remotely from the original infection. Aortitis

may be a legacy of hereditary syphilis. Thus may be explained many of the cases (which are nevertheless infrequently met with) of atheromatous disease of the aorta in young persons. In a young girl affected with syphilis, Virchow found the aorta studded with sclerotic and atheromatous patches. Hübner has described a nodular form of endarteritis thus produced. Baumgarten and Lancereaux consider that the disease commences in the external coat of the vessel (as a periarteritis). It is probable that the commencement of the disease may be in both these modes. I have noted how very varying are the degrees of suffering in cases of diseases of the aortic valves having syphilis as an antecedent. In some there may be little or no painful symptom, in others there is severe suffering, continuous or paroxysmal, graduating up to the agony of angina pectoris. It has seemed to me probable that in the former the internal and middle coats of the vessel are chiefly affected, while in the latter the inflammatory lesions affect the external coat and involve the nerve fibres and ganglia. Huchard has noted syphilitic antecedents in 35 cases out of 150 manifesting symptoms of angina pectoris.

Malaria may be a cause of chronic, as we have seen it to be of acute, aortitis; Lancereaux observed that in twenty cases in which there were found after death patches of aortitis in the ascending and transverse portions of the vessel, eleven had been the subjects of malarial infection.[4] It is probable that there are in many cases multiple causes for the manifestation of chronic aortitis and atheroma—for example, syphilitic infection and physical overstrain or gout and senility. In some cases the causes are difficult or impossible to discern. Though the affection is evidenced in the great majority after maturity and especially in old age it is seen sometimes in infants and children. Chronic aortitis causing obstruction of the aortic orifice has been seen in an infant of two months, and a patch of atheroma has been found in the aorta near one of the coronary arteries in a child less than two years old. Andral noted calcareous deposits in the aortic valves of a child of eight years.

Hollis notes that of 52 cases of atheroma no fewer than 35 had definite renal lesions, mostly of fibroid type, and in 28 there were pleuritic adhesions—he considers that a sclerotic process, a tendency to overgrowth of fibrous tissue in the body, is intimately associated with atheroma. Duplaix and Isnard had previously drawn attention to this sclerotic process as an important part of the changes about the wall of the vessel. The researches of many observers render it probable that the infiltration of tissues with the oval and round cells, nuclear bodies, is a direct agency in producing hypertrophy of the fibres of connective tissue.

My own observations have convinced me that the fibrous overgrowth is one of the earliest changes. While the endothelium is quite unaltered the fibres of the internal coat are seen to present wider meshes than in the normal and there is an evident fibrous hyperplasia. I believe those changes to be really secondary to lesions of the nutrient arteries of the vessel-wall and to the invasion of the deeper layers of the internal coat by the oval and round cells, though this is difficult to demonstrate. I believe that the elevations of the intima which are observed in the early stages of the disease are due to the imbibition of fluid in the widened meshes of the fibrous network and to the fibrous overgrowth.

Diagnosis.—In cases in which the disease, as in senility, has slowly progressed in the ascending portion of the aorta, well above the valves, even to extensive calcareous transformations, there may have been no declaratory symptoms. The diagnosis in such cases may be impossible during life; it is only made on the post-mortem table.

If the disease is in a limited patch above the valves, but not implicating the valve structures, diagnosis may be also impossible. It is only when it spreads to the immediate neighborhood of the coronary arteries and encroaches over their lumen that any sufficient signs may be manifested. These are dyspnœa on exertion, increasing feebleness and tendencies to fainting, the basic râles of œdema of the lungs, and the pretibial pitting of dropsy of the extremities.

In a large proportion of cases there are manifestations of precordial pain and distress at intervals—all these symptoms may occur in varying degrees graduating up to typical angina pectoris. It is to be remembered that in many cases not only are the orifices of the coronary arteries encroached upon by the patches of aortic atheroma, but the arteries themselves in some parts of their course are obstructed by additional atheromatous swellings in their own lining membrane. Jenner, of vaccination fame, first showed the frequent association of angina pectoris and blocking of the coronary arteries of the heart. Lussana discovered such obstruction in 21 out of 36 cases of the painful affection, and Sir John Forbes found the coronary arteries diseased in 16 out of 45 cases. Huchard has cited 145 post-mortem examinations in cases of angina pectoris in which disease involving the coronary arteries was demonstrated. In 64 cases both coronary arteries were implicated, in 37 the left, and in 15 the right coronary artery. There is, however, abundant evidence to show that angina pectoris in typical form may be manifested in cases where no morbid lesion of the coronary arteries is to be found, and many observers, like myself, have had post-mortem evidence of obstruction

and of calcification of the coronary arteries in cases in which no symptoms of angina pectoris have been manifested.

The important points in regard to practical diagnosis are these: When in a patient past the prime of life, or in a younger patient who has been the subject of syphilis, there are signs of difficulty of breathing on exertion (and perhaps of attacks of difficulty of breathing without assignable cause), and yet whose heart and vessels on physical examination present no signs of deviation from the normal, let the possibility of atheromatous involvement of the coronary arteries be present to the mind of the physician. Supposing that in such patient the distress should gradually but surely increase and râles be heard over the basic portions of the lungs, indicating pulmonary œdema, while there is as yet no physical evidence of valvular disease nor of dilatation of the chambers of the heart, the diagnosis of atheroma is rendered very probable. The occurrence of attacks of pain at the heart in paroxysms, as of angina pectoris, will greatly increase this probability, making the diagnosis almost certain; only it must be remembered that a differential diagnosis from a small aneurysm just above the semilunar valves may be impossible.

If the disease of the lining membrane of the aorta occurs in a small area just above the valves, but extending downward so as to encroach upon these, there will be the gradual development of the signs of obstruction, or insufficiency causing regurgitation, or the combined lesions at the aortic orifice. If the disease involves only a limited portion of the aorta and not the whole circumference, it will be found on careful physical examination that the great vessel is not dilated. The disease of the endarterium extending to the semilunar valves may cause these to be thickened in all their structures and their borders to unite so that the welded segments form a funnel, the smaller end pointing upward into the aorta. The thickened material may be firm, like cartilage, or of bony hardness from calcareous incrustation. Thus may be produced the condition of aortic stenosis. Far more frequently, however, the valves whose thickenings and hardenings afford an obstruction to the blood stream are also puckered and shortened so that they fail to close the orifice. Thus, in the great majority, there are both obstruction and insufficiency evidenced by the systolic and diastolic murmurs, while in a small minority there is obstruction only which is shown by a single systolic murmur. The late Dr. Fagge found, from an examination of the post-mortem records of Guy's Hospital, only two cases of pure stenosis to seventy-one of regurgitation. Supposing that the auscultation signs reveal a diseased condition of the aortic valves, the question now occurs, how shall we determine this as due to atheromatous changes

within the aorta encroaching upon the valve-segments? Roughly speaking, about half the cases we meet with are due to rheumatic endocarditis affecting the valve, and half to atheromatous disease within the vessel. The differentiation is of very high importance for practical therapeutics, and it is unscientific to rest content with the diagnosis of aortic valvular incompetence without attempting to ascertain the nature of the disease which has occasioned the valvular imperfection.

If the disease be due to rheumatic endocarditis the patient will probably disclose a history of an attack or several attacks of rheumatic fever. The murmur will be chiefly diastolic, heard over the aortic valves or down the sternum and toward the heart's apex; frequently it is diastolic only, but in some cases a systolic murmur is also heard over the aorta and the sternum. There will most probably be evidence of disease of the mitral valve, also the result of rheumatic endocarditis. The heart will be greatly enlarged, the carotid arteries pulsating and bounding, the brachials jerking, and the radial pulse sudden and collapsing—the typical Corrigan pulse. The face will be pale and the mucous surfaces anæmic.

If the valve disease be due to atheroma the patient, unless syphilitic or perhaps having the malarial cachexia, will be above forty years of age. There will be evidence of a gradual development of the disease, often unnoticed, without signs of acute or subacute rheumatism and presenting few symptoms until those characteristic of the aortic valvular imperfection are manifested. On auscultation two murmurs will be observed: the systolic, harsh and short; the diastolic, prolonged, aspirating, and inclined to be musical—or both murmurs may be harsh. At the mitral orifice there will be no murmur unless the orifice has become dilated from the intraventricular pressure; then the systolic murmur at the apex will not have the wide area of conduction which it manifests when the valve has been thickened by rheumatic endocarditis. The anæmia will be less marked than in the rheumatic disease, but the countenance may be yellowish. The degree of regurgitation, except in the latest stages of the slow process, will be inconsiderable; so the arteries, though tortuous and visibly moving or pulsating, will manifest less sudden pulsations. The temporals, the brachials, and the radials may present the hard outlines or firm plates hereafter to be mentioned. Examined by the sphygmograph there is not the tall, vertical upstroke with sudden and rapid fall as seen in the free regurgitation after the rheumatic disease, but the short or sloping upstroke of a toiling ventricle, the broad plâteau of an obstruction at the aortic orifice or within the arteries, and a less sudden fall in the descending line which is often broken by secondary vibrations.

If the atheromatous disease within the aorta exist over an extensive area so that the whole internal circumference is involved, careful examination will detect a general dilatation of the vessel. To investigate this the outline should be percussed out by means of a plexiimeter. The finger percussed upon will not give the true outline and evidence of the dilatation of the vessel may be lost.

By percussion with the finger of the right hand upon my plexiimeter (Fig. 34)[7] held between the forefinger and middle finger of the left hand, the process commencing well to the right of the sternum, an area where the sound vibrations begin to be modified in the sense of dulness can readily be mapped out and, the process being repeated to the left of the sternum, the outline of the dilated aorta can be delineated.[8] It is seen that this outline presents a uniform curve like the section, lengthwise, of a lemon or a pear. If it should present an irregular bulging from its border it will indicate saccular aneurysm; the even bulging tells of a fusiform or rounded general dilatation of the vessel—no pulsation will be felt as in aneurysm. In consequence of the dilatation of the aorta and the displacement upward of the innominate, the subclavian artery may be pushed upward so that it is felt to pulsate above the clavicle, and a thrill may be felt in this vessel and in the carotid. A sign, however, common to aneurysm of the arch of the aorta and the general fusiform dilatation will be observed on auscultation, viz., a loud, sudden second sound, often like a tap upon a small drum; sometimes it has a metallic clang. If the aortic valves are themselves diseased there may be no accentuation, but the second sound is muffled or replaced by the murmurs lately described. If the aorta has become so dilated that the semilunar valves no longer accurately close, the loud second sound is accompanied, or immediately followed, by a diastolic murmur.

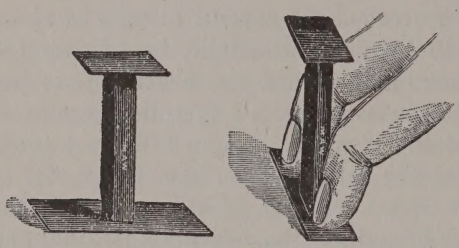

FIG. 34.—Sansom's Pleximeter, Made of Vulcanite. The second figure shows the mode of holding the pleximeter in adaptation to the part percussed.

The physical signs, therefore, indicate a general dilatation of the aorta above the valves and, if these be implicated, either an incompetence to close the orifice owing to their withdrawal toward the circumference or their actual disease through the extension into them of the atheromatous process as lately described. It is this form of the disease of the aorta to which Constantin Paul and some other French writers, with generous consideration for an English investi-

gator, have given the name Hodgson's disease (maladie de Hodgson), for Hodgson first in 1815 graphically described all the essential features. He gave an excellent account of the pulpy softenings of atheroma and the calcifications. He described the bulging of the aorta, commencing usually immediately above the semilunar valves, and the membranes of the arteries, thickened in a remarkable manner and incrusted with calcareous and atheromatous material, which formed the enveloping structure. He showed that the dilatation of the vessel in many cases ceased suddenly at the arch of the aorta, but that in others the normal calibre was gradually attained. He gave several examples of disease of the aortic valves consecutive to the affection which he thus described, and produced a complete picture of that pathological condition which, as I have said, shares in equal degree with the endocarditis occurring in association with rheumatism the pathogenesis of lesions of these valves.

Treatment.—The senile changes in the aorta, so long as they do not compromise the coronary circulation nor interfere with the due functioning of the semilunar valves, are usually unrecognized and therefore untreated.

If, though the signs of disease of the aortic valves are wanting, there are evidences of failing heart (as dyspnœa, faintnesses, œdema of the lungs, pretibial pitting and perhaps precordial pain or symptoms of angina pectoris) atheromatous blocking of the coronary arteries will be the diagnosis and the physician will counsel *rest.* There should be no half measures—the patient should be kept in bed, at any rate, for a week or two, the body being supported by pillows in the position which gives the maximum of comfort in breathing. In the next place I would advise an *all-milk dietary.* It may be a little difficult to persuade a patient past middle age—whose stomach has been the receptacle of foods of many and various kinds, far more than adequate to the needs of his organism, whose nerves of taste have been frequently and abnormally stimulated, and whose absorption of nutritive material and excretion of effete products have been in the nature of periodic and irregular thunder-showers which have deluged the land and blocked the drains—that he must come to the sweet simplicity which served for the earliest months of his life. Yet it is best. It is the absence of the irritation to the arterioles caused by the complex albuminoids which turns the balance toward amendment. It may be necessary, however, to make some concessions. In the early morning or on waking the patient may take half a pint of milk with half an ounce of rum or of cognac and an ounce of lime water. In some cases one to two ounces of fluid magnesia may be substituted with advantage for the aqua calcis. Three or four

hours afterward a second half-pint of milk may be taken flavored with a little hot coffee; the third half-pint, after a like interval, may be taken as a blanc mange made with isinglass or gelatin. At similar intervals during the remainder of the waking hours the changes may be rung with the various flavorings, but no solids should be permitted other than light biscuits (crackers).

The total amount of milk taken in the twenty-four hours should be three to six pints. The total quantity of cognac or spirits of any kind should be limited to two ounces. To reduce this and yet to break the monotony of the purely milk diet it is a good plan to have a firm jelly fully flavored with Madeira, rum, Kirschwasser, or Chartreuse. One or two tablespoonfuls of isinglass are to be melted in very hot water and the milk added thereto; the small quantity of gelatin thus mingled with the milk is sufficient to prevent any firm curdling of the casein in the stomach, the coagulum being rendered much softer and its digestion facilitated.

In regard to *medicinal treatment* the choice should, for the most part, be between the iodides, nitroglycerin (trinitrine), nitrite of amyl, and the alkaline bicarbonates. Of the iodides I think the most satisfactory is the sodium iodide in doses of 5 grains three times a day; the dose after some days of observation may be increased to 10 or 15 grains. For some periods the iodide of ammonium, of potassium, or of strontium may be substituted, the change being sometimes of obvious advantage. The iodides are arterial relaxants, and the freer circulation induced by such relaxation of the arteries is evidenced by greatly improved breathing, the relief of the general distress, and the cessation of pain and the signs of angina. Huchard has done excellent service in insisting on the value of the treatment of such cases by the iodides—a treatment which may be continued for months and even years. In some cases reported in his work[9] the treatment extended over a period of eighteen months and two years. To relieve dyspnœa at the moment, or for systematic treatment when the iodides, administered by way of the mouth, are ill-borne, the iodide of ethyl capsules, as in the treatment of acute aortitis already mentioned, may be employed for inhalation.

It is a necessary caution that the large doses of the iodides should not be persisted in for more than a week without interruption, and in case of the small doses there should be a withholding for one or two days at the end of each fortnight.

Trinitrine should be prescribed if there is any intolerance of the iodides or if these seem to be inefficacious. It may be given in one-minim doses of the one-per-cent. spirituous solution or in the form of tablets in which $\frac{1}{100}$ grain of nitroglycerin is combined with choco-

late. The dose may be administered every three or four hours and cautiously increased to five or even ten minims or the like number of tablets, *i.e.*, to $\frac{1}{20}$ or $\frac{1}{10}$ grain. For continued administration I prefer small doses ($\frac{1}{200}$ grain) three times a day, and I have seen very good results from this plan, so that all adverse symptoms have been kept in abeyance. A combination with nitrite of amyl is in some cases a distinct advantage as in the "tabellæ nitroglycerinæ" of the Westminster Hospital Pharmacopœia, which contain nitroglycerin $\frac{1}{100}$ grain, amyl nitrite gr. $\frac{1}{4}$, menthol gr. $\frac{1}{50}$, and capsicum gr. $\frac{1}{100}$ in each chocolate tablet.

A caution is necessary to patients who are entrusted with a prescription for nitroglycerin, for I am convinced that there may be a nitroglycerin mania as there is a morphinomania. A patient may take doses which are unnecessarily large at too frequent intervals even when the drug has acted to the full as an arterial relaxant.

The nitrite of amyl is most useful for inhalation at the time of crises, whether of difficulty of breathing or of anginoid pain, and it is best that even slight attacks when these occur suddenly should be thus treated. A paroxysm which might become severe is thus warded off and perhaps also the tendency to recurrence is reduced.

The glass capsules most commonly employed contain three minims of amyl nitrite; they are encased in cotton-wool and silk and, on their being broken, the liquid is liberated, soaking the wool and silk cover. The patient should inhale freely by deep inspirations, enclosing the broken capsule in the palms of both hands, which are apposed and applied as a hollow sphere over nose and mouth.

When there is evidence of lasting improvement a course of the alkaline bicarbonates may be substituted for the more pronounced arterial relaxants. Ten to twenty grains each of the bicarbonates of potassium and sodium may be given in solution three times a day for protracted periods.

A therapeutic agency of much value in these cases is *massage*. The effect of friction of the skin is a quickening of the rate of the heart beats. Muscle kneading, however, quickens the heart only in a minority of cases—usually it causes a slowing. Without doubt it greatly aids the venous circulation. The conclusion of Mr. A. Symons Eccles, who has made many observations with instruments of precision, is that after general kneading of the muscles the amount of blood delivered by each ventricular systole is increased and the force of such systole is augmented. "The effect of muscle kneading as shown by thermometric, sphygmographic, and manometric readings is to increase the force of the heart's action, the quantity of blood passing through the external organs (muscles and skin), and the

interchange between these tissues and the nutritive fluids, whose passage through them is thus facilitated" (*British Medical Journal*, December 1, 1888, p. 1,214). It seems that after abdominal kneading the heart's action becomes more slow and the volume of blood supplied to the external tissues, and probably the brain, is diminished, the greater amount of blood being retained in the vessels within the abdomen.

Massage, in the stages of treatment in which active effort is to be proscribed, supplies the place of muscular exercise. It should be applied chiefly to the muscles of the extremities and the thorax; the process should be continued from half an hour to an hour daily. Abdominal massage should be practised with caution and for only short periods.

Other important therapeutic questions are the action and the employment of *baths* and bathing. The effect of a hot bath is to relax the arterioles of the skin, to quicken the heart's action, and to enfeeble it so that the pulse of the wrist becomes very dicrotic. In fact the patient is weakened by bleeding into his own skin. The lassitude after a hot bath is a matter of common experience. A prolonged hot bath may be of great danger in the case of a patient with weak coronary circulation. A cold bath, on the other hand, contracting the skin arterioles, calls upon the heart for increased effort. If the heart be adequate to this call the rate of pulsations is reduced but each systole is more complete, there is a tonic effect upon the ventricles. If it be inadequate to respond, the effects are shock and arrest. The practical lesson, corroborated by much experience, is to this effect: Let the patient with weak coronary circulation be sponged daily with warm water, but at the finish let a cold or tepid sponging be given. A warm reaction soon comes and the general comfort is increased.

Although the policy of complete rest is to be advised in the early stages when the patient presenting signs of obstruction of the coronary circulation first comes under the physician's notice, it by no means follows that the forced idleness should be continued long. The endarteritis may be stayed; the circulation may become more free. A muscle which is in comparative disuse tends to waste. The heart should be, with caution, stimulated to increased physiological activity. Before the commencement of walking exercise, it is well that the patient, even while he is in the semi-recumbent position, be taught to make lifting movements of the arms in slow and regular manner. So the aspiration of the thorax is increased and circulation promoted. Gradually walking exercise may be permitted, sudden overstrain and undue fatigue being carefully avoided. The maxim to be observed is "hasten slowly."

The rigid rules of the dietary may now be relaxed, though milk should still form a large portion of the daily food. Fish, light meats, vegetables, and farinaceous puddings may now be partaken of, but an excess of albuminoids in the diet, all rich dishes, beers and strong wines, and alcohol in excess of the two ounces per diem (permitted as the maximum), must be prohibited.

It may be remarked that I have hitherto said nothing in favor of reputed *tonics*, general or cardiac. In a case presenting the symptoms which we are treating I am convinced that they do more harm than good. With the intent of increasing the strength of the debilitated patient and of improving the condition of apparent anæmia, iron has often been prescribed in these cases. The result is an increase of the tension within the arteries, already too great, a harmful consequence attained in like manner by the administration of strong beef-teas and solutions of meat extractives. The anæmia is often apparent, not real; the blood-corpuscles are not deficient in coloring matter but the arterioles are contracted and narrow. I have known of patients progressing very favorably under a course of iodides, or simply of alkalies, who have been tempted to "strengthen themselves" with iron tonics, with the result that after a week or two all the old symptoms of dyspnœa and cardiac distress have returned. The same is true, in many instances, of digitalis and cardiac tonics, the uses and the dangers of which we shall presently consider. Suffice it to say that in the treatment of cases now under consideration, digitalis, strophanthus, and caffeine are not without their dangers, but that a good effect may be maintained and adverse actions minimized by a combination of the cardiac tonic with an arterial relaxant as nitroglycerin. The value of the cardiac tonic in a given case is governed in greatest measure by the mechanical difficulties of the ventricles and the adequacy of the cardiac muscle. These circumstances we shall briefly review in coming to the consideration of a second class of cases in which the atheromatous disease has caused imperfections of the aortic valves.

This involvement may or may not cause a graver prognosis and increase the difficulties of treatment. A slight crumpling of the aortic valves by an atheromatous process which has become arrested may be far less serious than a small elevation which blocks the orifice or the lumen of a coronary artery. I have observed a case in which such incompetency of the aortic valves existed for more than fifteen years without adverse signs, and instances of still longer duration without symptoms are by no means uncommon. Compensatory hypertrophy of the left ventricle occurs to a sufficient degree to neutralize the effect of the leak in the valve and the balance of well-being

is restored. Yet it must be remembered that a twofold danger is present in such cases—the disease which involves the aortic cusp is perilously near the orifice of a coronary artery, and the train of symptoms we have just considered as indicating its obstruction may at any time be induced, or the imperfection of the valve may become greater than the heart muscle can overcome, and the symptoms of inadequacy of the ventricles are then manifested. We have already considered the former case, we now come to the latter.

We will take first the consideration of the condition in which the difficulty is mechanical—the valvular imperfection being too great for the ventricle to overcome. As I have said, this difficulty is less frequent than is the case of a valve rendered incompetent by the results of rheumatic endocarditis, but it is by no means uncommon in the atheromatous form. The intra-ventricular pressure is such that the cavity tends to dilate, and the mitral valve may be rendered inadequate to close its aperture. It is now that the arguments become more strong for the employment of digitalis and other tonics of the cardiac muscle, but it is well that we briefly estimate these *pro* and *con*.

To take the objections first. I have found that when regurgitation is very free and the left ventricle much hypertrophied, the administration of digitalis or digitalin may positively increase the distresses and the dangers of the patient by inducing sudden and violent contractions of the ventricle which may actually rupture an artery; —in other cases it may cause sickness, the patient being intolerant of the drug; in a third class it produces irregularity of the heart, causing coupled or linked beats and intermissions; in a fourth, though there may be apparent improvement for a considerable period, sudden death has occurred. My explanation of such a disaster is, that the drug has slowed the heart to a too great degree—the diastolic pause having been thus prolonged, the patient has bled from his carotids (and so from his cerebral arteries) into his own relaxed left ventricle, with the result of a fatal syncope.

Huchard has cited observations showing that distress and danger may be increased by the administration of digitalis to patients suffering from angina pectoris. He considers that the drug may be the determining cause of cerebral embolism in some cases of atheromatous disease. Traube brought evidence to show that it might bring about cerebral hemorrhages.

These observations should inculcate caution, but they by no means should induce us to withhold the drug from all patients manifesting atheromatous disease of the aorta. In the majority of cases of aortic disease due to atheroma, digitalis may be administered for

long periods. It may seem, if we look at the conditions superficially, that it is illogical when the evidence indicates high tension in the arterial vessels to administer an agent which shall augment the tension, but further consideration will show that theory consorts with practice. The danger in most cases is not so much the inordinate augmentation of ventricular force as the failure of the muscle of the ventricle to rise to the height of its requirements and thus to enter upon its retrograde course of degeneration. Its more perfect systole, as brought about by the cardiac tonic, means not only a better propulsion of blood through the systemic arteries but a better supply through the coronary arteries to its own structure. Careful observation for a few days will show whether the drug causes an undue violence of the systole of the ventricle. If so it must be at once withheld; so also if there be symptoms (such as vomiting) of intolerance. It should be omitted for at least a day at the end of each week of treatment. If the renal secretion which has been sufficiently free begins to become scanty, the digitalis must be withheld or another cardiac tonic substituted.

There may be differences of opinion as to the preparations of digitalis to be employed and the mode of administration. The tincture is perhaps the most frequently prescribed; some prefer the infusion, but this has no advantage over the powdered leaves, which necessarily contain all that can be extracted in the infusion and more. One grain of the powdered leaves is equivalent to a third of an ounce of the infusion and to eight minims of the tincture. The commencing dose of the tincture should be 5 minims three times a day; this can be increased to 10 and 20 minims, but after three to seven days' administration of the maximum dose it is best to omit for a like period. Where it is obvious that the continuous administration of a moderate dose is well borne it is a convenient plan to prescribe the powdered leaves in combination with alkalies: thus, bicarbonates of potassa and soda, of each 10 grains; powdered ginger, 5 grains; powdered leaves of digitalis, 1 grain; menthol, $\frac{1}{2}$ grain. The powder to be taken stirred in half a wineglassful of water three times a day.

Some physicians greatly prefer digitalin to digitalis. The granules of digitalin (Homolle) are much used in France, two, four, or five being administered in the twenty-four hours. Each granule contains $\frac{1}{6^{1}5}$ grain of amorphous digitalin. Huchard prefers a solution of *crystallized* digitalin in alcohol (with a small addition of distilled water and pure glycerin) of which fifty drops represent one milligramme ($\frac{1}{6^{1}5}$ grain) of crystallized digitalin. It is said that 1 grain of the crystallized is the equivalent of 4 grains of the amorphous digitalin. Huchard administers 30, 40, or 50 drops of the above solution. He

does not commence this medication until after a repose of a few days, during which there has been an all-milk diet, and after the administration of a purgative. He does not repeat the dose until after the lapse of fifteen or twenty days.

I have found in some cases digitalis quite without action, but the hypodermic injection of $\frac{1}{100}$ grain (exceptionally $\frac{1}{50}$) of digitalin has been attended by excellent results. This may be administered two days consecutively, but not re-administered for at least four days. I have always employed the gelatin discs (which I introduced for hypodermic medication in 1874) made by Savory & Moore. Each disc contains $\frac{1}{100}$ grain of digitalin; it is readily dissolved in two or three drops of water with the aid of heat (*Medical Times and Gazette*, 1874, p. 494).

The other cardiac tonics which may replace digitalis or may, in some cases, be administered with great advantage therewith, are caffeine and convallaria majalis. Citrate of caffeine may be best administered in 5-grain doses, with 1 fluidrachm of liquor ammoniæ acetatis and a sufficiency of water, three times a day for three days. Thus it acts as a cardiac stimulant and a diuretic. I have found the maximum effect to be manifested in some cases from one to three days after the cessation of the administration; the continued administration is sometimes attended by a diminution of the renal excretion. Convallaria seems to have none of the deleterious effects of digitalis nor of caffeine (for the latter occasionally induces insomnia). It may be administered as the fluid extract or the tincture in doses of from 10 to 30 minims three times a day. The administration may be continued for a fortnight without interruption; then it is best to suspend it for a few days. Though far less powerful as a cardiac stimulant than digitalis, I think it is much safer in aortic disease and often of much value. Strophanthus must be used with great caution in cases of aortic disease, and sulphate of spartein is not to be preferred to the broom tea (decoctum scoparii) which may be given with the other cardiac tonics.

It should be clearly understood that in disease of the aorta which is considered to be due to chronic aortitis, the treatment by digitalis or other cardiac tonics is not to be put in force as a matter of routine.

If the aortic valves are not involved but the coronary arteries are threatened, digitalis should be withheld until the treatment by rest, milk diet, and the arterial relaxants has been fairly tried.

If the aortic valves have been invaded, no agent which specifically alters the cardiac rhythm should be administered until the case has been observed for a sufficient period to convince the physician that compensation is failing and the ventricle is unequal to its work. It

is only when there are signs that the left ventricle is dilated or that the right chambers are being overloaded, or when the manifestations of dropsy or dyspnœa (as in mitral disease) indicate the failure of the ventricular muscle, that the treatment by digitalis, etc., should be commenced; and then a judicious stop should be put to the treatment when favorable symptoms show that compensation has been re-established. It is only in a very small minority of cases of aortic disease that benefit ensues from the protracted administration of digitalis in the manner adopted in a large number of cases of primary *mitral* insufficiency.

An important point in regard to treatment of cases of aortic atheroma is that we shall rightly estimate the condition of the heart muscle as influenced by other circumstances than those of the local disease. The heart of one patient is not as the heart of another. It follows that the system of Dr. A at B, or of Dr. C at D, cannot be confidently recommended to a given patient because he has atheromatous disease of the aorta or of its valves.

If, for example, such disease is declared in an obese person who has led a too sedentary life, there is a great danger that the heart-muscle may fail because it may suffer fatty infiltration and associated degeneration. Then the plan of treatment recommended by Oertel may be put in force. The essentials of this treatment are the limitation of fluids ingested, the diminution of fat-production by the administration of highly nitrogenized solids to the exclusion of fat, the promotion of vigorous efforts of the cardiac muscle by severe exercise of the body muscles in methodical and systematic hill climbing, and the fostering of free diaphoresis from the skin. The liquids absorbed are to be reduced to an equivalent of thirty-four to thirty-six ounces of water per diem, including that contained in the solids. The solids recommended as a daily ration are cooked meat, fish, chicken, or game, in all about twelve ounces, one or two eggs, a little salad or cheese and from four to seven ounces of fresh or cooked fruit, and not more than five ounces of bread. The ascents of hills or mountains are to be made gradually, by short steps, with stoppages, and the making of deep inspirations if dyspnœa comes on. It is expected that the exercise shall produce sweating, and this is considered to be salutary. Additional means for inducing a diaphoretic action, such as Turkish baths, may be used.

I am by no means able to recommend this method of treatment in the early stages of failure of compensation in aortic disease from atheroma even in an obese patient. I prefer a preliminary treatment by the milk diet and arterial relaxants, for I believe that the advance of the process of endarteritis presents the greater danger. The value of

the plan is much more decided in cases of mitral regurgitation in the absence of aortic disease. When, however, there has been such evidence of amendment that the physician is led to consider that the atheromatous process is not advancing, and muscular exercise becomes, as I have before said, a necessary therapeutic means, or when after one or two weeks of trial the plan of treatment by milk diet is not attended with improvement, the method of Oertel should be put in force in hope that the cardiac fibre may thus be restored to a more healthy tone.

Another plan of treatment which rivals Oertel's is that of the Drs. Schott at Nauheim in Hesse (Germany), not far from Frankfort. This consists essentially of the use of baths and methodical exercises of the voluntary muscles. One of the most important considerations in regard to the employment of the baths of Nauheim is their temperature. That of the water of the bath as administered to the patient is from 88° to 95° F.—that is to say, the bath is below the temperature of the blood—tepid or even cool. It is probable, therefore, that a tonic effect is produced upon the heart by the cool water, as I have lately explained. Another point which may be important is that the natural water of Nauheim as used in the bath contains free carbonic acid gas. There is evidence that the effect of this upon the sensitive nerves of the surface of the skin may be stimulation of the heart. The water contains also alkaline chlorides and salts of iron. The effect of the bath is usually to reduce the rate of the heart's contractions but to increase the force of the ventricular systole. The muscular exercises, which are a part of the plan of treatment, consist of movements of flexion and extension of the upper and lower extremities with rotations and flexions of the trunk, in orderly succession, each movement being, in a prescribed manner, resisted by an attendant so that the effort which is thus called forth is graduated by the degree of resistance imposed. The muscles being brought into increased physiological activity receive an increased blood-supply (their arterioles becoming dilated), the heart-beats are quickened by the reflex stimulus, and the ventricles are given an increased suction-power in diastole. Distention of the cardiac chambers is relieved and circulation becomes more free. If, however, the muscular efforts are too sudden and forcible, the arterioles are compressed by the contracting muscle fibres and an obstruction is caused to the blood-stream from the heart. In such case, when the walls are feeble or degenerated, the call upon the heart may be too great and it may fail, the result being a fatal syncope.

The details of the Nauheim treatment are fully described in a recent article by Dr. Thorne.[10] It has been affirmed that excellent

results have been obtained in the treatment of aortic disease and even of angina pectoris by this method. The value of the treatment in certain cases is no doubt great; but some words of caution seem to me necessary. A patient is apt to misunderstand the word "cure" as applied to the treatment at a foreign health resort and to invest the process there with a certain glamour. He prefers, it may be, the counsel of the prophet who bids him do some great thing to the advice of the friendly doctor who has carefully watched the signs of his disease. The physician may find that, though the signs of organic imperfection of the aortic valves remain, the cardiac muscle has become equal to the task of maintaining the balance of the circulation; but the patient, conscious that there is an abiding condition of disease, desires to attempt the impossible. He imagines that by the use of the mineralized baths the incrustations may melt away, as crystals in water. The doctrine that his strength is in quietness and confidence is not acceptable to him; but the fatigue of a long journey, the dislocation of the habits of his daily life, and the full stop put to his home comforts, may rudely convince him—but too late.

It is only by their effects, indirectly produced, upon the muscle of the heart that the methods just considered are beneficial. Yet in this relation they may be of high value. The senile heart of the subject of chronic disease of the aorta may be small and ill-nourished, and yet there may be no special signs of cardiac failure, only a generally depressed nutrition. Then a good dietary, with not too many restrictions and the hygienic plans described, are strongly to be recommended. In a patient with dilated heart who fails to improve sufficiently under treatment at home, the balance may be turned toward recovery by a course of treatment at one of the health resorts. Especially in a subject of chronic disease of the aorta threatened with fatty infiltration of the heart, the slave of self-indulgence and faulty diet, the prescribed routine and the kindly despotism of the doctor are therapeutic means of the highest value. The problem of causing the action of the heart to be in the greatest degree efficient is one present to the physician in all the moods of his patient and in all the phases of disease, and is not peculiar to the affections we have lately considered.

Aneurysm of the Aorta.

Definition.— A local bulging of the wall of the aorta, which forms a sac containing blood and constitutes a tumor that may press upon adjacent tissues.

Etymologically the word signifies a dilatation (*ἀνευρύνω*, to widen;

ἀνεύρυσμα, a widening), and has been usually applied to general as well as local dilatations of the vessel.

Aneurysms have been described as *true* and *false*. A *true* aneurysm, according to this classification, is a general dilatation, fusiform or globular, of all the coats of the vessel—the result of the chronic inflammation (atheromatous change) which has been lately considered. This fusiform dilatation is, as Dr. Douglas Powell[11] has said, "clinically speaking, *not* true aneurysm." This disease has a pathology all its own, and it is well that the term "true aneurysm" should cease to be applied to it. The saccular dilatations of the aorta, producing their effects in part by the morbid changes in and the distention of the coats of the aorta, and in part by pressure upon neighboring structures, have been termed *false* aneurysms. It is time that the term *false*, as thus hitherto applied, should be discarded.

Saccular aneurysms are termed *dissecting* aneurysms when, through a breach in the endarterium, the current of blood is forced between the coats of the vessel, tearing these and splitting them up to a variable extent along the length and breadth of the vessel; *varicose* or *anastomosing* when, by their increasing bulk and consequent pressure, they cause breaking down and absorption of a portion of one of the neighboring vessels so that there ensues a communication between the aortic aneurysm and the pulmonary artery, one of the venæ cavæ, or the innominate vein.

Morbid Anatomy.—At the commencement of the formation of an aneurysm, a portion of the middle coat of the aorta having become diseased, the support thus afforded to the blood-column is impaired; the force of the blood-stream therefore causes a bulging in the affected area. The internal coat is forced into the sacciform dilatation, and it may be traced for some little distance from the orifice by which this dilatation communicates with the healthy aorta on the internal surface of the sac, where it may be detected in detached patches. The external coat of the aorta forms the chief part of the bag of the aneurysm; its connective tissue increases, and the structures in immediate association with it undergo inflammatory changes. The fibrous tissue grows in and among the surrounding structures which, making common cause with the altered coats of the artery, constitute the external portion of the sac.

The opening by which the aneurysm communicates with the aorta may be wide or narrow. The late Dr. Sutton considered that the dangers were greater in aneurysms with wide openings. Aneurysms with narrow openings were more likely to be attended by bruits; an aneurysm over which no bruit is heard is, therefore, less ominous

than one attended with bruits. Often aneurysms are multiple; two or more are found in different portions of the aorta, and there may be extensive signs of atheroma and calcareous degeneration. In some cases, however, the aneurysm is seen in an otherwise healthy aorta. Of such Dr. Sutton said: "I have had a sense of awe on looking into the body and seeing that, while all the other organs and tissues were so exceedingly healthy, death had been caused by so limited a disease."

The sac may be found after death to contain only fluid blood, more commonly it is lined by layers of coagulated fibrin; the outermost layers are gray or whitish, sometimes pearly white, their blood-pigment having been removed; the innermost are dark red blood-stained. Frequently the blood has found its way between the layers, staining these in irregular streaks. There is no coagulation of blood in a general fusiform dilatation of the aorta, only in any subsidiary saccules springing therefrom or on projecting plates of calcareous material. The first stage of the formation of clot in saccular aneurysm is the adhering of white corpuscles to the internal, morbidly changed wall of the dilated vessel. These white corpuscles undergo a chemical alteration. The observations of the late Dr. Wooldridge tended to show that a substance called *lecithin*, which exists in the white blood corpuscles in union with fatty acids, was the determining agent of the coagulation of the fibrin. The leucocytes successively tending to the periphery, layer after layer of fibrin becomes deposited. Hammersten's explanation is that the coagulation is due to the action upon the fibrinogen of the blood of a ferment derived from a special variety of white blood corpuscles of small size (termed blood platelets); the ferment being only produced when these small corpuscles have undergone some disintegrating change. (See section on Diseases of the Veins).

The thoracic aorta, including the arch, is the artery of all others most liable to the formation of saccular aneurysms. Of 915 cases of aneurysm tabulated by the late Dr. Crisp, it was the seat in 382 instances—forty-one per cent. The abdominal aorta was the seat of aneurysm in 105 cases—eleven per cent.

Aneurysms situated just above the semilunar valves in the ascending portion of the aorta are apt to be very small; they are often rapidly fatal from rupture. Aneurysms of the aorta may vary in size from a pea to a child's head. An aneurysm is frequently met with at the junction of the innominate artery with the aorta, the aneurysmal sac being formed by both vessels. The arteries emerging from the aneurysmal pouch are in many instances obstructed; they may be narrowed by atheromatous change—or plugged by coagula—or the

growing aneurysm may drag upon them so as to reduce their aperture to a mere slit—or the enlarging sac may so press upon the arterial tube as almost to obliterate its lumen.

The effect of an aneurysm upon the heart is often to displace it but by no means necessarily to cause its hypertrophy or enlargement. A huge aneurysm may be accompanied by a small heart. Hypertrophy of the heart only occurs in sequence to aneurysm when the sac is so situated as to impair the integrity of the aortic valves. Eighty-two cases of aneurysm of the thoracic aorta, observed post mortem at the London Hospital, were tabulated for me by my colleague, Dr. F. J. Smith. Forty-one of these were in such positions that the aortic valves were *not* implicated; enlargement of the left ventricle was evidenced in only nine, and in three of these there was only slight hypertrophy. In forty-one cases in which the aortic valves were involved, hypertrophy and dilatation of the left ventricle were manifested in thirty-five.

Causes and Modes of Development.—Aneurysm of the aorta may be caused by direct injury. Instances of this mode of production are, however, rare, and it is difficult to exclude the probabilities or possibilities of predisposing forms of disease. The question of such mode of causation has come before a court of law, the case having been recorded by Dr. Grant of Melbourne. A gentleman, said to have been in perfect health, received a blow upon the chest in a railway collision, and from that time suffered from pain and dyspnœa. The signs of saccular aneurysm became manifest to the right of the sternum, and death occurred suddenly thirteen months subsequently. It was contended that pre-existing disease was not adequately excluded, but the verdict was in accordance with the view of the medical witnesses, who held that the sudden blow was the sole cause of the morbid changes resulting in the aneurysm, and heavy damages were recovered. It was unsatisfactory that no post-mortem examination was made. There have been many instances in which the aorta has been ruptured by violence to the external parts. The museum of the London Hospital contains specimens from four such cases. The rupture has been found in the external coat or in the middle and internal coats, or as a complete laceration of all the coats. It cannot be doubted that a blow upon the sternum and adjacent ribs and intercostal spaces is adequate in a healthy person to inflict such injury upon the coats of the aorta (without producing a complete laceration) as to cause subsequently an aneurysmal dilatation.

It may be considered more doubtful whether a severe muscular overstrain on the part of the individual can induce aortic aneurysm, the aorta at the time being perfectly healthy. It must be accepted

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as proved that persons whose occupations compel them to make powerful or sudden muscular efforts are liable to aneurysm of the aorta. The disease occurs especially in porters, dock laborers, oarsmen, and such mechanics as lift heavy weights or make inordinate movements of their arms. It has been observed that in the United States immigrants who have to do exceptionally hard work for a livelihood are far more liable to aneurysms than the home population. Of 242 fatal cases in New York during nine years, 81 were in persons born in the United States and 161 in immigrants (Hirsch). It is in the case of soldiers, especially those in the British army, that the lesson has been in the highest degree enforced. Lawson found aneurysms to be eleven times more frequent among soldiers than among members of the civil population.

Meyers concluded from extensive observations that the majority of aneurysms in the English army were due to the severe exertion of the soldier when encumbered by heavy knapsacks and accoutrements, and hampered by tight clothing. Parkes confirmed this opinion. Sibson stated that aneurysms were chiefly situated in those portions of the aorta which during severe bodily exertion were subjected to the greatest strain. The statistics of the army corroborated this view. In 109 cases the situations were, ascending aorta 37 cases, arch 38, descending aorta 12, thoracic aorta 7, abdominal aorta 15. Those of Karl Malmsten, of Stockholm, gave in 101 cases, arch of the aorta 57, descending aorta 17, and abdominal aorta 4. In some cases the origin of an aneurysm at the moment of a severe or sudden muscular effort has been indicated by symptoms—as by something having cracked or given way. It is very difficult to realize, however, that the increased blood-pressure within a perfectly healthy aorta brought about by even the severest muscular exertion could give rise to the local bulging constituting a saccular aneurysm. It would seem more probable that the whole vessel would dilate. Yet there might be with great probability a bursting of a small artery supplying the coats of the aorta—one of the vasa vasorum—the effect of which would be an aortitis and softening of the deep layers of the internal and the adjacent middle coats. In the case in which the processes were slow and the proliferation of fibrous tissue abundant, there would be the ordinary signs of chronic aortitis (atheroma). If the softening predominated and the support of the fibrous tissue of the external coat were insufficient, aneurysm would result. In the case of the manifestation of the immediate symptoms of pain and distress, there would probably have been a softened area produced by ruptures from previous muscular overstrain. Although the event may be comparatively rare, there is a very considerable probability that a softening of

the middle coat initiating a saccular aneurysm may occur from overstrain in a previously healthy aorta.

In a large proportion of observed cases aortic aneurysms are produced by the causes already considered, which give rise to subacute or chronic aortitis. At the post-mortem examination the aneurysm or aneurysms are seen in an aorta showing often very extensively the signs of atheroma and calcareous change. Any of the causes—gout, alcoholism, malaria, syphilis, besides overstrain and the degeneration of old age—may induce a limited aortitis which may result in softening of the wall of the vessel. The augmentation of pressure within the aorta by even a slight muscular effort would determine the aneurysmal dilatation.

It is shown from the facts, however, that the proclivity to aneurysm is not parallel with that to atheroma of the aorta. We must, therefore, agree that the factors of atheroma operate unequally in the production of aneurysm, and this view is confirmed by further investigation. "While deterioration of the arterial coats, as typified in atheroma, is most common after the age of sixty, one of its ordinary consequences, aneurysm, belongs to an earlier period of life" (Sir Richard Quain). Isolated saccular aneurysms may occur in quite young men and women, though aortic aneurysm is more common in males than females in the proportion of about 8 to 1. Of 92 cases analzyed by the late Dr. Hayden, 60 occurred between the ages of thirty and fifty.

As I have said, one cause, though probably an infrequent one, of the disease in the earlier periods, or in the prime, of life may be muscular overstrain alone. The cause, however, which operates in the most pronounced degree is *syphilis*. Prostitutes suffer much more from aortic aneurysm than do other women. Aitken found that at least fifty per cent. of aortic aneurysms occurred in soldiers with syphilitic infection and with no other ascertainable morbid conditions present. Karl Malmsten (of Stockholm) stated that in about eighty per cent. of the cases collated by himself the existence of syphilis was definitely made out. In about five per cent. only, gout or rheumatism were the antecedents, and in five per cent. alcoholism could be assigned as a cause. According to this observer the chronic aortitis due to syphilis may have a special form termed by himself "aortitis sclero-gummosa." I have observed many cases of aortic aneurysm in which the origin from syphilitic disease of the aorta has been evident. This disease in some cases seems to be of the nature of a gumma like those which occasionally are found in the muscular walls of the heart, and in others has the ordinary characters of chronic aortitis. The course of such disease may be subtle, unattended with symptoms, and

tragically fatal, or it may give rise to phenomena which simulate other diseases and make diagnosis difficult or impossible. A preparation in the London Hospital Museum shows a ruptured small aneurysm of the aorta, one of several aneurysmal dilatations about the commencement of the arch and the ascending portion within the pericardium. The rupture had taken place into the pericardial sac. There were many erosions of the internal coat of the arch of the aorta, which was much dilated, and smooth elevations of the lining membrane to the left of the orifice of the left subclavian artery. These changes were found in the body of a prostitute, aged about 30, and were no doubt syphilitic; they had progressed undetected, for the woman died suddenly while at breakfast and there had been no previous symptoms. In another case observed by me, that of a married woman, aged 29, who was infected after her second marriage (three years before her death) there was a large aneurysm of the abdominal aorta which had displaced forward the left kidney, had caused erosion of the eleventh and twelfth dorsal and first lumbar vertebræ, had perforated the diaphragm, bursting into the left pleural cavity, and had also ruptured into the left psoas muscle. Small opaque patches of disease of the lining membrane of the aorta were also seen in the ascending portion of the vessel, and a depressed, puckered cicatrix (showing a thinning of the coats) just below the mouth of the aneurysm, which measured one inch by one inch and three-quarters, in the abdominal aorta. The pain from which this patient suffered was not ascribed to its true cause, but was thought to be a form of neuritis following an attack of influenza. It was strangely variable, being almost absent during the day, but severe in the night. The extensive disease within the vessel had not been betrayed by symptoms, and even the destruction and displacement of organs by the great aneurysmal tumor was not attended by continuous pain, and the patient was able to walk.

The conclusions which I have formed are that aneurysms of the aorta may be caused by external violence, by internal strain induced by severe or violent efforts—the initial lesion in such cases being a rupture of some branches of the vasa vasorum,—by chronic aortitis due to various morbid influences—a softening of the middle coat in such case being the predisposing, and muscular effort the determining cause,—and by syphilis, in which case the initial softening of the middle coat may occur without symptoms and the aneurysmal dilatation may be effected without the determining agency of any unusual muscular effort.

Course and Terminations.—An aneurysm of the aorta may burst and cause death by the thinning of the coats of its sac. This may

occur in the case of a very small aneurysm of the size of a pea or a cherry. The rupture not infrequently occurs into the pericardium. Or by its increase in size the sac may cause pressure on the surrounding parts and consequent necrosis of these; in such cases rupture may take place into the cavities of the heart, as the right auricle (a rare occurrence), into the superior vena cava (of which Pepper and Griffith have collected twenty-nine cases), into the pulmonary artery, the trachea or bronchi (the latter more frequently; our museum possesses five such specimens), into the œsophagus, into the peritoneum or retro-peritoneal tissues, or into the intestines, not infrequently the duodenum. More rarely the aneurysm points at the external surface and bursts through the skin. The rupture of an aortic aneurysm constitutes the most sudden form of sudden death. I have seen the body of a man who died from this cause in the act of getting into bed. Death must have been instantaneous, for he was in the position of drawing the bed covering over him, one leg remaining uncovered. His son, who was sleeping at his side, awoke to find him dead.

The increasing bulk of the aneurysm may produce suffering and death by pressure on neighboring parts without rupture. Compressing the superior vena cava, it causes engorgement of the veins of the head and upper extremities. If it compresses the inferior vena cava, the results are œdema of the lower extremities and ascites. The sac of an aortic aneurysm may press upon the pulmonary artery; it may then induce phthisis pulmonalis, just as in cases of stenosis of the pulmonary artery, which almost invariably ends in tuberculosis of the lungs. If the sac presses against the trachea, it produces cough and urgent dyspnœa, while pressure upon a bronchus may be evidenced by bronchorrhœa, bronchiectasis, and suppuration in the lung—"a process," says Osler, "which by no means infrequently causes death in aneurysm, and a condition which, at the Montreal General Hospital, we were in the habit of terming aneurysmal phthisis" ("Principles and Practice of Medicine," Edinburgh and London, Pentland, p. 679). Involvement of the recurrent laryngeal nerve, usually the left, in the sac may cause hoarseness, loss of voice, and laryngeal stridor. Compression of the œsophagus may occur and cause dysphagia, and contribute to death by starvation. In rare cases the thoracic duct has been compressed. Pressure backward on the vertebræ causes their erosion and necrosis; the spinal canal may thus be opened and the cord compressed.

An aneurysm may rupture, at any period during the course of its enlargement, not suddenly into a cavity, when the outpour of blood is abundant and rapid, but by a slower process of oozing into sur-

rounding cellular or parenchymatous structures. It is then termed a *diffuse* aneurysm. The effused blood sets up some adhesive inflammation of the parts adjacent. The transverse portion of the arch and the abdominal aorta are the situations from which saccular dilatations, tending to produce diffuse aneurysms, chiefly arise. The resulting cellulitis is a painful complication.

An aortic aneurysm may cause hæmoptysis by (*a*) compressing the trachea and causing congestion and rupture of the vessels of its mucous lining; in this case the spitting of blood is not profuse but the expectorated mucus is blood-stained; (*b*) by breaking down of lung following the invasion of the aneurysmal sac into the lung tissue; or (*c*) by rupture into the trachea or bronchi. The possibility of an aneurysm being thus the cause of hæmoptysis should be present to the mind of every physician. The amount of blood expectorated at the commencement of the rupture of the aneurysm may be slight; but some hours later the quantity may be appalling, and the patient may die in a few seconds.

The investigation of a case of hæmoptysis coming before the physician for the first time should be made with much gentleness, and the complete repose of the patient should be enjoined until the evidence is sufficiently positive to indicate the source of the hemorrhage.

In dissecting aneurysm the blood escapes between the internal and middle coats of the vessel and separates these for a variable distance. Sometimes there are two apertures; one the rent by which the blood originally entered, and the other at a situation more remote from the heart, where it has penetrated through the internal coat and rejoined the aortic stream. In some cases the formation of a dissecting aneurysm does not aggravate the distresses and dangers of the ordinary saccular form of the disease. A case is recorded of a soldier who had aortic aneurysm for more than twenty-nine years; at the post-mortem examination there was found a dissecting aneurysm which formed a double tube round the aorta in its whole length. The rupture of a dissecting aneurysm may, however, cause sudden death.

In some cases of aortic aneurysm there have been manifestations of angina pectoris. These generally indicate aneurysms at the root of the aorta. The pain in many instances radiates down the left arm or up the neck and to the throat; sometimes it is in the course of the upper intercostal nerves. It seems probable that in these cases there has been a neuritis of the cardiac plexus, the irritation having been propagated from the diseased external coat of the aorta adjoining the plexus. Intense pain with epileptiform symptoms may occur in the bursting of an aneurysm.

In the case of a man, aged 38, observed by me, who presented the signs of a large aneurysm of the abdominal aorta, there had been attacks of sudden and severe pain for three months, referred to the epigastrium and both hypochondria; these were paroxysmal and periodic, but were provoked by exertion. In addition there was an almost constant pain in the back. Under rest and treatment the pains became relieved, then, after an action of the bowels, the man was found in an epileptic fit. When the convulsion subsided there was active delirium, requiring three men to restrain the violence of the patient. On palpation over the site of the aneurysmal tumor, I found that the force of pulsation formerly manifested had greatly diminished, the slightest pressure causing signs of great pain. I commenced the administration of chloroform by inhalation, and the patient soon became calm. In half an hour the signs of extreme distress recurred, and chloroform was administered at intervals throughout the night. On recovery from the chloroform-sleep there were indications of intense pain. In the early morning a dose of forty minims of liquor morphiæ acetatis failed to give any relief, and the chloroform inhalation was continued, though at longer intervals than previously, until 2 P.M., when a hypodermic injection of one-third grain of hydrochlorate of morphine produced a protracted calm. The inhalation of chloroform, at first with short and afterward with longer intermissions, had been continued for nearly twenty-six hours. The following day there was a threatening of a return of the paroxysm, but calm was induced by the hypodermic injection of half a grain of hydrochlorate of morphine. Freedom from pain was maintained till six days subsequently, when the face suddenly blanched, and death took place two hours afterward. At the post-mortem examination an aneurysmal tumor of the size of two oranges in juxtaposition was seen to involve the abdominal aorta below the origin of the superior mesenteric artery; it had burst between the layers of the peritoneum in the transverse and ascending meso-colon as far as the cellular tissue of the pelvis. The bodies of the last dorsal and two first lumbar vertebræ had been much eroded, the intervertebral disks standing out from the remains of the vertebræ nearly half an inch. The appearances were such as to point strongly to the conclusion that the commencement of the rupture of the aneurysmal sac was coincident with the epileptic paroxysm and with the severe pain with which it was associated. The treatment adopted no doubt not only relieved the pain but checked the outflow of blood from the ruptured sac.

Spontaneous cure may take place in aortic aneurysms. The cavity of the sac becomes filled with layers of fibrin until the dimensions of the interior of the vessel are restored. The outermost layers of fibrin, together with the external coat of the sac of the aneurysm, become shrunken, dense, hard, and in some cases infiltrated with lime salts.

Diagnosis.—In the case of an aneurysm situated just above the valves there may be no symptoms, and sudden death may occur from its rupture, a diagnosis having been impossible. It has been

observed by Hayden and Nixon that aneurysms in this situation tend to descend in their progress of growth; "by their position they are sheltered from direct influx from the ventricle while they are exposed to the maximum force of reflux from the aorta." On the other hand aneurysms well above the valves are subject to distention by the force of the direct current issuing from the left ventricle. If the sac be in such position that the coronary artery is obstructed, the symptoms would be identical with those, already described, of block of the coronary from atheroma. A differential diagnosis could not be made, but the question is of little importance, for treatment should be the same in both cases. In the case in which a small aneurysm causes imperfection of the aortic valves, giving rise to a diastolic murmur or to systolic and diastolic murmurs, there may be an impossibility of the differential diagnosis from disease of the aortic valves due to atheroma. It is only when the sac of the aneurysm presses upon surrounding parts or begins to cause a visible prominence of the wall of the chest that there can be any precision in diagnosis.

Aneurysm of the Ascending Portion of the Arch.

An aneurysm of the first or ascending portion of the arch of the aorta is to be recognized by one or more of the following points: (*a*) Bulging forward, the sac may form a tumor in which pulsation may be detected usually but not invariably. The situation of the prominence is generally to the right of the sternum in the second or third intercostal space, more rarely in the corresponding space on the left side of the sternum. The sternum itself may contribute to the prominence; the bone may become eroded by the continued pressure of the sac and ultimately perforated, the wall of the aneurysm at the perforated area being then constituted by the inflamed subcutaneous tissues and the skin. The tumor may be painful and tender, any pain referred to it being aggravated by exertion. In some cases the hand placed over it detects a thrill as well as a pulsation, and occasionally a systolic murmur (very rarely a diastolic) may be heard over its situation. (*b*) Growing toward the right, the aneurysmal tumor will compress the superior vena cava, causing venous engorgement and œdema of the upper extremities. It may burst into the superior vena cava, causing the signs to be mentioned. (*c*) Expanding upward, it compresses the upper lobe of the right lung or the right bronchus, producing at first impeded breath sounds, afterward dulness on percussion from consolidation of the lung. It may also compress or disorganize the right recurrent laryngeal nerve. (*d*) Pressing toward

the left, the sac obstructs the pulmonary artery, tending to produce not only the signs of phthisis pulmonalis, already mentioned, but also dilatation of the right chambers of the heart. Possibly the sac may rupture into the pulmonary artery, which accident may be indicated by sudden and urgent dyspnœa with hæmoptysis and rapid death. (*e*) By its increasing bulk the sac may displace the heart, causing its apex to be below and to the left of the normal; and (*f*) very rarely it may compress the inferior vena cava, causing œdema of the feet and ascites.

In the *inspection* of the patient, care should be taken to arrange the incidence of light, or else slight pulsations may be overlooked. The uncovered chest should be well illuminated, and the observer should bring down his eyes nearly to the level of the patient's chest-front, if the subject be recumbent; or if the observation be made in the sitting or erect position of the patient, the eyes should view the chest in profile; thus slight pulsations are seen. The movement of an aneurysmal sac with each systole of the heart is expansile. This is sometimes to be demonstrated by a method described by Fagge and Pye-Smith. The observed prominence is covered by a piece of adhesive plaster, which has a slit cut down its centre; if this be narrowly watched, the slit will be seen to widen with each expansile pulsation; or the method may be adopted of affixing light paper columns or cones of cotton-wool to opposite points of the pulsating surface, and observing whether or no these diverge; if the tumor be expansile, they are seen to separate as radii. Two stethoscopes may be held, applied by their chest pieces and their divergence demonstrated. Careful inspection may show pulsation in the intercostal spaces to the right of the sternum, or in those of the upper portion of the chest on the left; the pulsation may be so strong as to lift the sternum and the ribs. Investigation shows that this pulsation is expansile, thus differing from the throbbing observed in cases of general dilatation of the aorta. Over the site of the aneurysmal tumor a prominence may be observed—a prominence not correlative with the evidence of pulsation; in fact, such elevation may be seen when there is no pulsation, the reason being that thick deposits of fibrin within the sac prevent the pulsatile expansions. Over the prominent skin surface the venules may in some cases be seen dilated and varicose. In late stages, when the tumor nearly approaches the surface, the overlying skin may be very thin, or there may be subcutaneous inflammatory changes.

Palpation confirms and extends the evidence obtained from inspection. A pulsation which is communicated to the upper part of the sternum is nearly always due to the aneurysm of the thoracic aorta in its ascending or transverse portions. Such pulsation may be felt

to the right of the sternum. It will generally be found that the impulse given to the hand is not so sudden as it is heaving and expansile.

From a series of 32 cases of aneurysm of the thoracic aorta observed at the London Hospital, in which pulsations were detected, I have found that in 25 (about eighty per cent.) these were to the right of the sternum and in only 6 (less than twenty per cent.) to the left thereof. The situation of maximum pulsation was the second right intercostal space in 11 cases; in 5 additional cases the pulsation was to the right of the sternum but not precisely localized; in 9 other instances the pulsations were in various sites from the sixth rib below to the right clavicle above; in one case only was there a central pulsation of the sternum rising into the neck. In some instances a thrill may be felt, in some also the period of the second sound of the heart is marked by a shock felt by the hand or the fingers. This shock is usually accompanied by a sound, and is best appreciated by the ear applied to the chest, with little intervening covering, or by the use of the wooden stethoscope. The shock is due to the falling back of an unusual column of blood—the normal, plus the quantity contained in the aneurysmal cavity—against the aortic valves. It is an important confirmatory sign where other evidences of aneurism are present.

Percussion in the investigation of a case of suspected aortic aneurysm is best done with the aid of a pleximeter. The outlines, when the fingers alone are employed, are less precise. I have in many cases mapped out a bulging of the vessel when percussion by the fingers only has given no sign. In a considerable number of cases such evidence has been obtained when no pulsation whatever has been observed. Care must be taken to detect the pulsation if present; light should be directed upon the upper portion of the præcordium, so that a slight movement may be observed and its situation marked, and palpation must be employed with care. The outline of the heart in relation to that of the bulging vessel must be noted. In cases of aneurysm of the arch in which the aortic valves have been unaffected, I have found that the outline of the heart, as determined by percussion, is not large, but often rather small. On the other hand, when the aneurysm is so situated that the aortic valves have been rendered incompetent, signs of hypertrophy of the left ventricle are observed. The position of the heart is sometimes altered by the aneurysmal tumor, and the percussion outline indicates such change of situation. As the aneurysm increases in bulk, its outline may be demonstrated to the right of the sternum, from the first rib to the upper border of the liver dulness, for the tumor may expand until its extension downward is stayed by the diaphragm and subjacent liver.

Auscultation may indicate a murmur over the aorta or over the prominence suspected to be an aneurysm, but it must be remembered that in many cases of saccular aneurysm murmurs are not present. Douglas Powell has said that in about half the cases of sacculated intra-thoracic aneurysm which came under his notice no murmur was to be heard. In a considerable proportion there was no murmur audible to the end of the case. Of 132 cases taken from the records of the London Hospital in which the diagnosis of aortic aneurysm within the thorax was made, only 12 were distinctly stated to present systolic murmurs alone, and of these only 5 were over the situation of the ascending aorta or the aneurismal tumor in this position. In 25 the systolic were accompanied by diastolic murmurs, and in 6 diastolic murmurs alone were heard.

I have found valuable auscultatory evidence in some cases of aneurysm, when a murmur has been absent, or very feebly heard, over the thorax, by causing the patient to place within his mouth the small chest-piece of the binaural stethoscope, and to close his lips over it. On auscultating thus, the observer may hear a distinct or loud systolic murmur in the case of a thoracic aneurysm, the vibrations being communicated to the trachea, and thence directly by the air-column to the ears.

In some cases a diastolic murmur may be heard at greater or less distance from the aortic valves in cases of saccular aneurysm of the aorta. If this be in the ascending part of the arch, thoroughly clear of the valves, the second sound may be heard sharp, loud, and uncomplicated; while above, near the right sterno-clavicular junction, a diastolic murmur, entirely separate from the heart sounds, may be evident. This is due to the elastic recoil of the aneurysmal sac after its ventricular systolic distention and is of course coincident with the diastole of the ventricles. Most frequently the murmur is double—systolic as the blood rushes in through the roughened borders of the pouch, diastolic as it is impelled back—but I have heard it diastolic only.

If there be a loud continuous murmur, manifesting increase of loudness with each cardiac systole, accompanied by thrill, but heard independently of the normal sounds of the heart and only over those parts where the aorta is in close relation with a great venous trunk, this must be considered as evidence of a communication between artery and vein—a varicose aneurysm of the aorta. If the maximum of such sound be about the second intercostal space it is probable that there is a communication between the aorta and the superior vena cava or with the right auricle.

In the absence of murmurs, altered characters of the second

sound constitute very important evidence. As in the general dilatation of the aorta this sound may be accentuated, ringing, clanging, or drumming; but, more than this, the ear may be conscious that it has a sort of double character. Palpation may already have convinced us that there is a diastolic shock or jog. The ear is more capable than the finger of appreciating this sign, for it both hears and feels at the same time. The shock of the second sound is heard, and the elastic recoil of the aneurysmal sac is felt. I think the best mode of investigating it is by the ear direct, with only a slight intervening chest-covering. An exaggerated aortic second sound is not to be regarded as an invariable sign of aneurysm of the aorta within the thorax. The sound may be accompanied by, or be replaced by, a murmur, or the valves may be thickened and the sound of their closure rendered dull. We must therefore look for other signs to aid our diagnosis.

Pressure Signs.—The aneurysmal sac may form no prominence anteriorly and none of the signs we have considered may be detected, but it may produce very pronounced symptoms by pressure in other directions. When an aneurysmal tumor causes pressure upon the superior vena cava or both innominate veins, the venous channels at the root of the neck may form spongy masses immediately above the clavicles, and those of the head, neck, arms, and upper part of the chest may be observed to be much distended. The eyeballs may become prominent, and the arms, together with the head and neck, may be swollen from œdema. Bristowe has shown that even in cases of complete obstruction of the superior cava the resulting venous blockage and œdema may be to some extent averted or remedied by the establishment of vicarious channels. These routes are sometimes demonstrated as bulky and tortuous veins in unusual situations, as from the axilla to the groin, a junction being formed with the external iliac (Bristowe: "A Treatise on the Theory and Practice of Medicine," 5th edition. London: Smith, Elder & Co., 1884, p. 551). Byrom Bramwell has said that aneurysmal tumors are less likely than solid intra-thoracic growths to produce obstruction to the venous return.

Such venous distention and œdema in the upper part of the body may also be brought about by any tumor capable of causing compression of the vena cava, by thrombosis in the vessel, by bands of cicatricial tissue, the result of inflammation constricting it from without, or by rupture of an aneurism of the aorta into the vessel. Confirmatory signs of aneurysm, therefore, must be sought for.

The occurrence of a rupture of an aortic aneurysm into the superior vena cava is characterized by a sudden onset of severe symptoms.

In some cases the obstruction to venous return by the pressure of the aneurysmal sac has been betrayed by the signs just described; in others the onset has been quite sudden with no previous symptoms. Intense venous congestion of the upper part of the body is the rapidly developed visible sign. The lividity and œdema may be manifested in the head and arms and not in the thorax; this depends on the site of the perforation or compression. If the obstruction be above the azygos vein, the lividity and œdema are confined to the head and arms; if it be below the point of entrance of the azygos, the chest will share in the congestion. When the thorax is involved there is sometimes a sharp line of demarcation between the upper affected and the lower unaffected parts of the body, marked by numerous small varicose veins around the lower part of the thorax. In some cases a greater œdema is manifested on one side; a long standing pressure upon, and obliteration of, the left innominate vein may allow the establishment of a collateral circulation. In such case the sudden rupture of an aneurysm may induce œdema only of the right side. If the rupture occur in such position that the blood current is directed into the right innominate vein, there will be lividity and swelling of the right arm. If the communication be, not with the vena cava, but with the right or left innominate vein, there will be a corresponding unilateral œdema in the upper portion of the body. There is dyspnœa, intense in some cases, slight or nearly absent in others. Ewart describes it as especially of the form of expiratory dyspnœa in one case. Hæmoptysis also may occur. The other signs of aneurysm, pulsating tumor, thrill, abnormal outline of percussion, dulness (especially below the right clavicle and under the upper portion of the sternum), murmur of continuous character in the case of varicose aneurysm, etc., must be diligently sought for (see valuable paper by Pepper and Griffith in *The International Medical Journal*, October, 1890).

Aneurysm of the Transverse Portion of the Arch.

An aneurysm in the transverse portion of the arch of the aorta may cause pressure upon the trachea and induce stridor and severe dyspnœa. Paroxysms of stridulous breathing and suffocative cough may be manifested. Pressure may be exerted, not upon the trachea, but on the left bronchus, and this will be shown by deficient respiratory murmur and reduced expansion of the left lung. Pressure on the left recurrent laryngeal nerve produces the signs of enfeeblement of the left vocal cord—very rarely both cords—and the resulting symptoms. Pressure on the œsophagus may cause dysphagia and the usual symptoms of œsophageal stricture; these signs are of much

importance in diagnosis. Pressure on the branches of the sympathetic nerve and the involvement of its fibres in the aneurysmal sac may cause inequality of the pupils; usually the pupil of the affected side is contracted, rarely dilated, in comparison with its fellow. Pressure toward the front of the thorax causes bulging or necrosis of the tissues between the aneurysmal sac and the surface of the chest. A tumor pulsating in the position of the innominate artery may be due to an aneurysm of this vessel, or to a saccular dilatation of the aorta. The presence of tracheal tugging and accentuated aortic second sound or diastolic shock will indicate that it is aortic. Pressure on, or interference with, the innominate or left subclavian artery produces its effect upon the pulses of the side affected.

Among the *general evidences* of thoracic aneurysm are progressive enfeeblement and emaciation, with signs of exhaustion from the pain, and symptoms of distress. Lancinating intercostal neuralgia is a symptom which calls for a diligent search for signs of aortic aneurysm on the part of the physician. Sudden pain of this character may occur in the left chest, unattended with the symptoms of angina pectoris, several times a day with intervals of perfect ease. "Such a pain," says Dr. J. Chris. Lange, "which does not yield to treatment should always excite a suspicion of, and determine an examination for, aneurysm" (*International Clinics*, January, 1892).

An important physical sign of aneurysm of the thoracic aorta in the portion of the arch under consideration is that known as *tracheal tugging*. The following are the methods by which this symptom should be investigated: (1.) Place the patient in the erect position, direct him to close his mouth and elevate his chin to the fullest extent; then grasp the cricoid cartilage between your finger and thumb and exert gentle upward pressure. If aneurysm exist, the pulsation of the aorta will be distinctly felt transmitted through the trachea to your fingers. The act of examination will increase laryngeal distress, should this accompany the disease (Surgeon-Major Oliver's method). (2.) The observer stands behind the seated patient whose head is slightly thrown back and steadied against the observer's chest. The tips of both index fingers are then inserted under the lower edge of the cricoid cartilage which is gently raised by them (Dr. William Ewart's method). The sensation imparted to the fingers by these methods of investigation is not a pulsation but a traction—a tugging downward—felt with each beat of the heart. Pulsation may be transmitted from the carotids, but tugging is peculiar to aneurysm. In some cases, the movement of the larynx is so distinct that the pulse may be counted by placing the tip of the finger upon the pomum Adami and making slight upward pressure. The sign is produced only

when the aneurysm is so situated as to press from above downward upon the left bronchus, or upon the portion of the trachea which is contiguous to it. It is not observed when the innominate artery alone is the seat of aneurysm. It may be evidenced when the other physical signs of thoracic aneurysm are absent and it is sometimes a very early sign in the history of a case.

Laryngoscopic examination is of very great importance. In any case in which there is presented the slightest probability of aneurysm of the thoracic aorta—and we know how obscure the signs are in many cases—there should be the routine employment of the laryngoscope. A loud, clanging, brassy cough should give rise to a suspicion of aneurysm; or a strident sound of the voice, imperfectly under the control of the will, sometimes assuming a falsetto character; or a more or less enfeeblement of the voice to a whisper. In some cases the voice is shrill and a crowing noise may be emitted on any muscular exertion. Yet there may be none of these signs, nor any symptoms suggesting a morbid condition of the larynx, and nevertheless an altered state of the vocal cords may be discovered on laryngoscopic examination.

On examination the observer may see that in ordinary inspiration there is little if any difference in the position of the two vocal cords; the left may be a little nearer the median line. The left capitulum Santorini and the left aryteno-epiglottidean fold may be on a somewhat higher level than their fellows of the opposite side. On phonation, the patient being asked to make the sounds softly of "ah" and "ay," the left vocal cord may be seen to remain fixed, while the right advances to the median line; or the right vocal cord may be seen to advance to the middle line and project beyond it. It may encroach so far as to meet the flaccid left cord, the cartilages overlapping when a high note is sounded. Thus, while the whole of the right cord is in view, only a portion, about half or one-third, of the left cord can be seen.

The cause of these signs is a paralytic lesion of the left recurrent laryngeal nerve. In the local bulgings of the wall of the aorta, the nerve, as it loops behind the arch of the vessel, becomes implicated. The mechanical irritation may produce in it inflammatory changes —neuritis. Thus, with the concurrent irritation propagated to the vagus nerve itself, may be explained some of the sudden paroxysmal attacks of urgent dyspnœa to which certain of the subjects of thoracic aneurysm are liable.

An instance which enforces the importance of the laryngeal signs has been communicated to me. A gentleman who presented very slight and obscure symptoms and no physical signs of aortic aneu-

rysm, consulted two physicians in London whose opinion is of high value. They both found slight weakness of the left vocal cord, and expressed their views of the probability of aneurysm and the necessity of care on the part of the patient. An injudicious acquaintance persuaded him that all such fears were groundless, and he undertook an expedition which involved considerable physical exertion and fatigue; death from the rupture of a small aneurysm of the aorta took place six months after the laryngeal signs were first noticed.

The *eyes* should be examined and careful observation should be made of the size of each pupil. In aneurysm of the thoracic aorta the pupils may differ strikingly, the left being usually contracted. This is chiefly in consequence of the destruction of the nerve elements involved in the aneurysmal sac, causing a paralytic lesion of the ciliospinal branches of the sympathetic; there is a paralysis of the dilator muscle of the iris supplied by the sympathetic, and consequently an unopposed action of the sphincter of the pupil supplied by the third nerve. It must not be forgotten that inequality of the pupils is occasionally congenital, that the effect may be produced by intrathoracic growths other than aneurysms, and that the appearances may be simulated by adhesions of the iris occurring exclusively in the one globe, or in one to a greater degree than in the other. The difference between the pupils, especially when the left is observed to be the smaller, is an important confirmatory sign of aneurysm.

One of the most important signs in the diagnosis of aneurysm is afforded by the *palpation of the arteries* on the distal side of the supposed saccular dilatation and comparison with the corresponding arteries of the opposite side of the body. It is better to commence the investigation by observing the vessel from the site of the supposed aneurysm in its course toward the periphery than to pursue the converse plan, which is often adopted, of comparing first the peripheral arteries (the radials, for example).

If an expansile pulsation to the right of the sternum indicates an aneurysm of the first part of the aorta, there will be no difference to be detected between the pulsations of the arteries of one side and those of the other unless the innominate is involved in or affected by the aneurysmal swelling.

If, with or without any palpation evidence of pulsatile tumor to the right of the sternum, communicating its pulsations to the sternum or palpable in the episternal notch, there are signs that the arteries of the right side present a diminished volume, while those of the left are normal, there is a primâ-facie case for aneurysm of the aorta, involving the innominate artery, or aneurysm of the innominate artery itself.

The observer should compare the pulsations of the carotid on each side, and the characters of the walls of each artery. If he note an enfeeblement of pulsation or diminution in calibre of the right carotid, he will proceed to compare the brachials. In the case of aneurysm, the like comparative deficiencies will be manifest in the right brachial. Next he will carefully observe the radials, and note the enfeeblement or obliteration of the right as a confirmatory sign of aneurysm affecting the innominate artery or its origin from the aorta.

The observer should also carefully determine whether, on the one side or the other, there is any delay in the radial pulse. He should palpate the two radials simultaneously, first in the ordinary position and next when he has elevated the patient's arms to the fullest extent. To exclude false impressions, he should change hands, so that he feels each artery with his own right and left fingers consecutively. Again, he should palpate the radial pulse of each side in relation with the apex-beat of the heart or the pulse of the carotid. If he note that, while the radial pulse of the left side is synchronous with the apex-beat and the carotid pulsation, that of the right side is delayed (or comparatively feeble and delayed), the evidence of aneurysm involving the innominate or subclavian of the right side is very strong. The diagnosis would point to a subclavian aneurysm alone if the right carotid were unaffected and the other signs of subclavian aneurysm, lately mentioned, were present. It must be remembered that in aortic aneurysm affecting the innominate the subclavian and carotid arteries may be unequally affected, so that there may be comparatively more pulsation in the latter than in the former, or *vice versa*.

If it be observed that, while normal characters and pulsations are manifested in the arteries of the right upper extremity and neck, the left carotid, left brachial, and left radial arteries present deficiencies of calibre and of pulsation, aneurysm probably involves a portion of the arch of the aorta and implicates the left carotid and subclavian arteries. In rare cases the left carotid alone may be thus affected. When a small aneurysm involves the left subclavian alone, the observation of diminished and delayed pulse in the left brachial and radial arteries may be the only sign of the disease.

The following are the modes in which an aneurysm modifies the pulse in a distal artery: An aneurysmal sac intervening between the heart and an artery may (1) act as an elastic reservoir and, reducing the pulsatile flow, make the expansion and collapse of the artery more gradual. The pulse as felt by the finger will be much diminished in force and suddenness as well as, perhaps, in calibre—there is only a gradual rise and fall felt in the vessel; or the consequence

may be (2) an obstruction to the distal artery, its mouth being narrowed by the encroachment of the fibrinous coagulum lining the aneurysmal sac. In such case the signs observed are diminution in the calibre of the vessel and enfeeblement of the pulse; or (3) the outermost coat of the aneurysmal sac may press upon the artery, obstructing its channel or even obliterating it. In this case there is diminution of the calibre of the vessel so that it may be detected with great difficulty—or degrees of enfeeblement ensue to absolute extinction of pulsation. The compression of vessels giving rise to the signs mentioned may be due to other tumors than aneurysm, and occasionally to endarteritis or other morbid changes; the lumen of the innominate, the carotid, or the subclavian artery may be obstructed and signs similar to these just considered are produced.

The signs observed from careful palpation of all the available arteries on the distal side of a supposed aneurysm—and I strongly recommend that the palpation be from centre to periphery—are often more valuable than those obtained by the sphygmographic exploration of the two radials.

The *sphygmographic examination* of the two radial arteries is valuable, however, as a means of record, and it has proved in some cases of much importance in diagnosis. Care must be taken that the tracings of the pulses of the two sides are taken under such precisely similar conditions that they may be susceptible of strict comparison, one with the other. Under similar degrees of pressure the vessels may yet be unequally affected, because the intervening tissues between them and the button of the lever may differ on the two sides. A very slight variation in the adaptation of the pad to the artery may produce a slight difference, which may be too readily accepted as evidence. In health and disease the sphygmograms of the right and left artery, obtained by the exercise of great care and under conditions which seem to be identical, may vary considerably. In certain cases the sphygmographic signs of difference between the two radial pulses in thoracic aneurysm are decided and incontestable. One radial artery may furnish a tracing, showing all the elements of the normal, while the other may present a mere rise and fall, having none of the normal elements of the sphygmogram; or there may be a tracing with sloping upstroke, rounded apex, and obliteration of secondary curves. If the tracing of the right radial has the aneurysmal characters noted above, while that of the left radial presents the elements of the normal trace, it shows that an aneurysmal sac intervenes between the left ventricle and the right radial artery—that is to say, the dilatation of the wall of the vessel must be in the ascending aorta, involving the innominate artery, in the innominate

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artery itself, in the subclavian, or in the brachial or radial. If the conditions are reversed, and the right radial presents normal while the left shows abnormal characteristics, then there is evidence that the ascending part of the arch and the origin of the innominate artery are not involved in the aneurysmal dilatation, while the left subclavian is so involved or there is an aneurysm in some part of the arterial trunk, on the cardiac side of the left radial. When the sphygmographic trace presents on both sides abnormal (aneurysmal) characters, the indication is that the aorta, from the origin of the innominate artery as far as that of the left subclavian at least, is involved in the aneurysmal dilatation, or else that separate saccules exist at those parts of the vessel whence these arteries emerge.

Aneurysm of the Descending Portion of the Arch.

An aneurysm of the descending portion of the arch of the aorta is characterized in many instances by a severe fixed aching or boring pain near the spine in one interscapular region or on both sides. The pain is due to erosion of the vertebræ. There may be also pains radiating round the chest from intercostal neuralgia. A pulsating tumor may be felt between the scapulæ or the body of the bone may be forced backward or perforated. The necrosis of the vertebræ continuing, there may be disease of the spinal cord with paralytic and other symptoms. The sac may press on the œsophagus, causing obstruction, and may eventually burst therein. Such rupture may occur in the case of a small aneurysm producing no symptoms and may cause sudden death. More frequently an aneurysm in this situation bursts into the pleura. The sac may press upon the left bronchus, producing at first obstruction to the air entering into the left lung (causing enfeeblement of respiratory murmurs as compared with that of the right), then fibrous thickening evidenced by bronchial breathing and bronchophony; there may be the signs of pneumonia, profuse bronchitis, or phthisical destruction of lung. Osler has noted as a sign of large thoracic aneurysm an obliteration of the pulse in the abdominal aorta and its branches. In a case in which no trace of pulse could be felt in the abdominal aorta, in the femoral, and in the peripheral arteries of the leg, a large area of pulsation was discovered in the left scapular region. The great sac of the aneurysm probably acted as a reservoir to such extent as to annul all pulsatile action in the arterial vessels on its distal side.

Differential Diagnosis of Thoracic Aneurysm.—The states of disease most likely to be mistaken for aneurysm of the thoracic aorta are (*a*) pulsating vessels or pulsations communicated to other structures by them; (*b*) solid tumors.

A pulsation may be observed to the right of the sternum in some cases when the lung has been drawn away from the aorta by adhesion. In the case of aortic regurgitant disease coexisting, the pulsation would be more marked and the diagnosis more difficult; an instance of this conjunction has been recorded by Osler. The absence of the defined excursion of percussion dulness which would indicate a local bulging of the aorta, the finding that the pulsation was not expansile, and the positive evidence concerning morbid changes in the lung and pleura, would lead to a correct diagnosis.

Pulsation to the left of the sternum may be due to uncovering of the pulmonary artery by fibroid disease of the lung. I have seen a well-marked example of this. Aortic aneurysm was considered to be negatived by the absence of pressure signs and of accentuated aortic second sound and jog—the diagnosis was proved correct by the post-mortem examination.

Pulsation to the left of the sternum occurs in rare cases of emphysema, and may be observed over a limited area, so simulating aneurysm of the aorta. There are always signs of pulmonary phthisis; the pulsation is not expansile and of course the pressure signs of aneurysm are absent (Comby, *Archives Générales de Médecine*, 11 and 12, 1883).

It is to be remembered that pulsation of the aorta may be visible in some nervous diseases such as exophthalmic goitre. The aorta may be so displaced as to beat forcibly to the right of the sternum in cases of spinal curvature.

Solid tumors within the chest may cause the pressure signs of aneurysm and in some cases the difficulties of diagnosis may be great, though usually a correct opinion may be formed by attention to the rules aready given. Malignant growths are most likely to cause these difficulties. These are usually primary and originate in the bronchial glands or the remains of the thymus; when secondary they occur as nodules in the pulmonary tissue; the discovery of a primary cancer or sarcoma in some other part of the body will, of course, aid the diagnosis. Cancer of the œsophagus producing pressure on the left bronchus may simulate aneurysm of the transverse or descending portions of the arch of the aorta.

Aneurysm of the Abdominal Aorta.

The most frequent site from which an aneurysm of the abdominal aorta springs is the part of the vessel just below the diaphragm and above the orifice of the superior mesenteric artery.

If the aneurysmal sac bulge forward, a pulsation below the ensiform cartilage, usually somewhat to the left of the mesial line, may

be detected on inspection. Palpation will confirm this evidence or perhaps detect a pulsation which is not evident to the eye. If a pulsation be felt at the epigastrium in the median line, it must be carefully noted whether there is an expansile swelling of the abdominal aorta, or whether the pulsation is communicated only by the vessel itself. A very forcible throbbing may be felt which may give the idea that there is aneurysm of the abdominal aorta; but the diagnosis of saccular aneurysm must not be given hurriedly, for pulsations from nervous causes are very common, while abdominal aneurysm is rare. As Sir William Jenner has said, "Instead of being your first, it should be your last idea that abdominal pulsation is due to aneurysm." In the majority of abdominal pulsations there is no structural alteration in the vessel, but an excited throbbing of the abdominal aorta, due to vaso-motor derangements. The phenomenon is most evident in people of spare build, in cases of anæmia, and in dyspepsia —especially in the dyspepsia of old people whose arteries have commenced to degenerate. In such the palpitation of the abdominal aorta may be a serious cause of discomfort. This condition may be differentiated from aneurysm, because in the latter there is a localized swelling of the vessel occurring more to one side, which, with the systole of the heart, expands equally in all directions, as may be readily manifest, when the whole hand is made to grasp the vessel. A tumor superficial to the aorta may pulsate from the communicated impulse of the vessel, but then no lateral pulsation will be felt. To aid in the diagnosis, the patient may be caused to bend forward on the hands and knees; a tumor isolated from the vessel recedes from it in this position, and the pulsation is not felt, while pulsation of aneurysm is not affected. It is said that epigastric pulsation caused by aneurysm of the lower part of the thoracic or upper part of the abdominal aorta is much influenced by respiration, that it is strong during expiration, but may even disappear during inspiration. In vaso-motor and nervous affections the epigastric pulse is quite as marked during inspiration as during expiration—it may be even more marked when a deep inspiration is taken. In some cases the diagnosis between pulsation of the abdominal aorta from nerve causes and aneurysmal dilatation of the vessel is very difficult. It may be legitimate to place the patient under an anæsthetic and explore. The pulsatory abdominal aorta is due, as Douglas Powell has said,[11] to vaso-motor disturbance, the tonus of the vessel being relaxed. The affection is allied to the goitrous disturbance of innervation of the thoracic aorta, and may be brought about by hæmic, emotional, malarial, and reflex causes.

A murmur systolic in time may be heard over the site of pulsation,

but it is of little diagnostic importance, as it is found in cases of neurosal pulsation. In a few cases a diastolic has been found to follow the systolic murmur—this is conclusive of aneurysm.

In certain cases there are no signs of pulsation, the sac of the aneurysm pointing laterally and posteriorly. If the tumor is high up beneath the pillar of the diaphragm, it may attain very large dimensions without being evident to palpation. It is surprising how large an aneurysm and how considerable a displacement of organs may be thus undetected when the sac is close to the vertebral column and does not bulge forward. The diagnosis must then rest on the subjective symptoms of the patient. Chief of these is pain, which may be continuous or intermittent. (*a*) The continuous pain is generally of a boring or gnawing character, referred to the back, sometimes fixed at a certain spot over the vertebræ. This pain may be aggravated by pressure or by stamping movements on the part of the patient and relieved by the recumbent position with the back upward. Such pain indicates erosion of the vertebræ. The erosion continuing, there may be invasion of the spinal cord causing numbnesses and tinglings, and finally paraplegia. (*b*) The intermitting pain may radiate with sudden violence through the abdomen and extend upward to the chest or downward to the inguinal regions and the testicles; it may be severe in the back, and may encircle the body as a girdle. It may last for a few hours (rarely longer than three) and suddenly cease, leaving the patient exhausted. This form of pain is compatible with erosion of the vertebræ. I have seen a case in which an aneurysm of the abdominal aorta had caused extensive necrosis of the eleventh and twelfth dorsal and first lumbar vertebræ, and yet the pain was not constant but chiefly nocturnal. During the day, for a long period, the patient was free from pain; subsequently the suffering came on in paroxysms lasting about two hours.

In some cases a heaving pulsation may be felt at the back over the ribs and interspaces contiguous to the dorsal and lumbar vertebræ and a prominence may be visible. A murmur may be heard to the left of the spinal column and, in this position, will be a decided indication of aneurysm.

The aneurysmal sac may obstruct or displace some of the abdominal organs. Compressing the common bile-duct it may induce jaundice. The left kidney may be pushed forward. The sac may extend backward and upward, pressing against the diaphragm or perforating it and bursting into the left pleural cavity.

The cœliac axis and its branches may be involved in an aneurysm of the abdominal aorta. Isolated saccular aneurysms may spring from the splenic artery. Those of the superior mesenteric artery are

very rare. A few cases (ten or twelve) of aneurysm of the hepatic artery are on record. Small aneurysms of the renal artery are more frequent; these may rupture, causing retro-peritoneal hemorrhage.

Treatment.—The diagnosis of aneurysm in any part of the aorta having been made, it is of the first importance that the patient should be enjoined to *rest.* Recumbency in such position as affords the greatest comfort should be the rule at the early periods of observation of the case. How this rule may be relaxed at later periods of the history will be considered hereafter. Repose is necessary for two reasons—first to lessen the distention of the sac and consequent growth of the aneurysm and the tendency to rupture caused by the augmented contractions of the ventricle excited by movements of the body; secondly, to favor the deposition of layers of fibrin within the sac by tranquillizing the blood-movement therein. The quietude resulting from the absence of all muscular exertion may cause a reduction in the heart pulsations of some thousands a day and convert a forcible injection into an almost equable flow.

The regulation of the *diet* of the patient is a matter of much importance. Valsalva first insisted upon a very restricted dietary and practised frequent venesections; Bellingham advocated starvation without the bleedings; Tufnell systematized the plan so that the treatment by enforced rest with restricted and regulated diet is now generally known as the Tufnell method. The patient is allowed for breakfast 2 ounces of white bread and butter and 2 ounces of milk or cream; for dinner 3 ounces of meat with 3 ounces of potatoes or bread and 4 ounces of water or claret; for supper 2 ounces of bread and butter and 2 ounces of milk or tea. The totals of food ingested in the twenty-four hours would thus be solids 10 ounces, liquids 8 ounces. Tufnell reported ten cases of aortic aneurysm cured by this combined plan of complete muscular rest and restricted diet. The essential objects are to reduce the volume of fluid in the circulation and to make the blood proportionately richer in fibrin. Of course there being very little expenditure of muscular force, much less than the average amount of food consumed by a healthy man is necessary. It is very difficult, however, to make a patient observe such ascetic abstinence for the period recommended of eight to thirteen weeks. I have advised the reduction of the fluid (which should be milk) to one pint in the twenty-four hours, thirst being relieved by the sucking of small pieces of ice; the maximum amount of meat or chicken allowed to be 6 ounces with 3 ounces of potatoes or green vegetables and a small amount of toasted bread or rusks (2 ounces).

With regard to medicinal treatment the most important is that by *iodide of potassium* as recommended by Bouillaud and Balfour.

The commencing dose should be 5 grains three times a day; after three days or a week this should be increased to 10 grains, and later to 15 or 20. The drug must be omitted if there are any signs of iodism, as running at the eyes and nose, frontal headache, diarrhœa, or the appearance of purpuric eruptions. I have continued the administration of iodide of potassium in cases of aortic aneurysm for many months and even for years (with occasional omissions), and I am convinced of the value of the treatment.

The foregoing should constitute the routine management of any case of aortic aneurysm, whether the sac presents toward the surface or not, when the patient first comes under the notice of the physician.

In the case of a sac—whether shown by an excursion of the aorta dulness or by a visible or palpable tumor—presenting in the front of the chest, or one at the back near the vertebral column attended with painful symptoms, I advise the systematic application of ice-bags or the local use of *cold* by the means of Leiter's tubes. The ice-bag should be suspended from a cradle so that a part of its weight only shall rest over the situation of the aneurysm, the rest of the body of the patient being comfortably covered. In cases in which there is much local suffering there should be renewed applications so that the local cooling is practically continuous night and day. In others, the applications may be made for two or three hours with intervals of half an hour or an hour; these intervals being lengthened after two or three weeks. The cold applications have a great influence in mitigating and often abolishing the suffering, and I feel sure that they aid in reducing the size of the sac. Very probably they tend to increase the tendency to fibrin deposition. I am of opinion that such employment of measures of local cooling can materially shorten the period during which absolute rest is to be enforced. If there are decided signs of amendment at the end of four or six weeks, the patient may be allowed to leave his bed for some hours each day, but he should return to the recumbency for another two weeks when there may be again some graduated exercise. Then will come the question whether or no any ordinary avocations are to be allowed. In most cases such permission may be given. Much good, conscientious work has been done by those who have been the subjects of aortic aneurysm, and the very protracted treatment by recumbency is a grievous burden often greatly resented. Two months of such treatment will induce consolidation within the sac if such consolidation is within the possibilities of therapeutics. It is to be remembered that though we may hope for a shrinking of the tumor in the great majority of cases we can expect nothing like a disappearance of it; it is still a

tumor, though if filled up with hard fibrin it is an almost harmless, inert thing.

It is only in the case of aneurysm with small openings from the aorta that we can hope for that which is tantamount to a real cure. In those with wide openings—though in all the treatment must be in hope, for it is time only that can determine the question of curability—all treatment, save that which is palliative is unavailing, unless some of the surgical proceedings soon to be considered be adopted.

As adjuvants to the treatment hitherto advised, I would mention (1) *aconite*, which is of much value when there is distress from forcible pulsation. Small doses (one to five minims) of the tincture suffice. One or two minims in water may be administered every hour during the continuance of palpitations or pain, to be omitted as soon as the phase of suffering has passed; then larger doses (four or five minims) may be given every four hours if there be a tendency to recurrence. (2) Hypodermic injections of *morphine hydrochlorate* (gr. $\frac{1}{4}$ to gr. $\frac{1}{2}$) may be necessary in circumstances of extreme pain. The employment of the ice-bags or cold applications, however, has greatly reduced the necessity of employing aconite or morphine. (3) *Phenacetin* in gr. v.–x. doses, preferably in cachets, or antipyrin (gr. xv.–xx.) in solution, may be administered with great advantage for the minor forms of pain, especially when symptoms of migraine complicate the case. (4) *The abstraction of blood*, by leeches or venesection, is a valuable method, giving great relief when there is much venous turgescence or when attacks of dyspnœa are distressing features in a case. It is scarcely necessary nowadays to inculcate caution in the use of this therapeutic method; indeed, it is probably too infrequently adopted as a means of relief. (5) *Ergot of rye* in doses of from twenty minims to one fluidrachm of liquor ergotæ ammoniatus well diluted, administered three times a day, was considered by the late Dr. Sibson to cause shrinkage of the sac. Sir William Broadbent has noted a case in which this treatment was followed by a diminution of the volume of the aneurysm (shown by casts of the tumor as it presented itself at the external surface taken before and after the treatment) as well as by a very marked abatement in the pulsation. Ergotin has been used for hypodermic injection by Langenbeck. Acetate of lead has also been employed, but there is no reliable evidence of its efficacy.

If after two months of treatment by rest and the means indicated, there are no signs of increase of the sac and no complications of moment, the patient may be permitted to do some muscular exercise, always remembering that he should avoid sudden overstrain. The patient must be watched, however, and must return to the treatment

by recumbency if there are symptoms indicating increasing bulk of the sac. In common with many observers I have seen the subjects of aortic aneurysm who have lived a life of usefulness and comfort for many years in spite of their malady. Sir William Broadbent has observed patients who remained well for ten years; Dr. Ord has mentioned a similar case. Hayden said that the duration of life in aneurysm of the abdominal aorta is from fifteen days to eleven years. No doubt in a small minority life has been protracted a much longer time, for we meet with cases occasionally, as I have stated, in which cure has taken place and the aneurysm is discovered after death from other causes.

If the tumor project in the front of the chest, a mechanical support, carefully modelled to its configuration and lined with soft leather, may be a source of comfort and an advantage to the patient.

In the case, however, of increasing bulk of the tumor or of evidences of greater encroachment upon neighboring organs, some of the more special methods of treatment—methods which are not to be undertaken without due appreciation of their risks and dangers—are to be considered.

Distal Ligature of Arteries.—For aneurysm of the innominate artery, the aorta contiguous thereto, or the transverse portion of the arch, deligation of the right carotid artery, of this artery and the right subclavian, or of the left carotid artery has been practised. Simultaneous occlusion of the right carotid and subclavian arteries by aseptic ligatures has now been performed in about ten cases. The most successful operation was that performed by Mr. Christopher Heath, the patient surviving four years. Death, however, has occurred in some cases in a few hours. Ligature of the left carotid artery seems to be a less formidable proceeding and one that has given better results. A man operated on in this manner by Mr. Heath lived four years; benefit has been recorded in several other instances. The *modus operandi* of the distal ligature is difficult to realize, but it is probable, as Mr. Heath says, that the alteration of the blood-current within the aneurysm effected by the ligature of the artery beyond it leads to a deposition of laminated fibrin within the sac. It must not be forgotten that even if this good result be obtained, the cure is not certain; for though the enlargement of the aneurysm may be arrested, the solid fibrin-containing sac may still produce pressure effects, and blood from the arterial stream may find its way between the layers of laminated coagulum and burst at some point of the circumference into the surrounding tissues.

Introduction of Inert Substances into the Sac; Filipuncture.—This method of treatment was first practised by Mr. Charles Hewett Moore

in 1864. He introduced twenty-six yards of fine iron wire into an aortic aneurysm; the result of the operation was the formation of a fibrinous coagulum within the sac, but the patient died from septic poisoning. Various substances have been chosen by subsequent operators for introduction within the sac to determine, as foreign bodies, the coagulation of fibrin and to serve as a meshwork in which the coagula should be entangled—horse-hair, catgut, watch springs, steel wire, and silvered copper wire have all been employed. Of nine cases of aortic aneurysm and two of innominate so treated, only one has had an encouraging result—an abdominal aneurysm treated by Loreta in which twenty-two yards of silvered copper wire were conveyed into the sac after abdominal section. The aneurysm became consolidated and reduced in bulk to the size of a walnut. The patient was discharged apparently cured on the seventeenth day. He died, however, on the ninety-second day after the operation, suddenly from a rupture of the sac at its junction with the aorta. The teaching of recorded cases seems to be that there is great danger in the performance of the operation of introducing irritating or septic agencies with the supposed inert filiform substance—that a silvered wire is the best material hitherto employed—but that the method is one that is condemned by the logic of facts. A plan which is much more hopeful is Macewen's method in which metallic needles are introduced into the sac for limited periods and then withdrawn. A successful case is recorded by Bignone (*Riforma Medica*, March 1st, 1895).

A man, aged 68, was admitted into hospital with well-marked signs of aneurysm of the ascending thoracic aorta, a pulsating, expansile tumor being evident in the third intercostal space near the sternum. Two steel needles were introduced into the sac of the aneurysm, were allowed to remain for twenty-four hours and were then withdrawn. A similar treatment was practised on three successive occasions within ten days. Satisfactory results appeared from the first, the pulsation after the fourth operation almost disappeared, and the tumor became reduced to one-third its original size. A fifth puncture was made three weeks after the fourth, and then the needle seemed to pass through a fibrous mass to reach a small cavity in the centre. The patient was now able to sit up all day and walk about without any pain or discomfort.

Galvano-puncture.—Petit has stated that the method of electrolysis for the purpose of obtaining fibrinous coagulation within the sac in cases of aneurysm of the aorta has been adopted in 114 cases, and of these 68 were improved. A case recorded by Ciniselli thus treated was observed sixty-six days after the operation and declared to be completely cured. In another case described by McCall Anderson the operation was several times repeated; the patient, a woman aged

46, progressively improved and seven months after the commencement of treatment the external tumor was reduced to about one-quarter its former size and was felt to be solid and only slightly pulsating. The patient said she felt in perfect health, experiencing only a sense of slight pulsation within the chest.

Galvano-puncture, in my opinion, should be attempted only in the case of thoracic aneurysms which are prominent at the surface, after treatment by the means previously described has proved futile, when pulsation is forcible and when the absence of murmur tends to indicate a large opening in communication with the aorta.

The operation should be done with antiseptic precautions. A battery of twelve Leclanché or ten Stöhrer cells is required. In most cases the patient should be carefully anæsthetized. Two needles should be inserted directly through the skin covering the tumor into separate portions of the sac. The communication should then be made between the needles and the battery, the current being gradually increased to a strength of from twenty to thirty milliampères. The positive pole (anode) may then be moved about (as Dr. John Duncan has advised) within the sac so as to touch and slightly cauterize the points of the internal surface of the sac within its reach. After from ten to twenty minutes the current should be gradually reduced and then disconnection made. The positive needle should be the first to be withdrawn; if difficulty be experienced in this, the current should be reapplied in a reverse direction until the needle becomes loosened from the firm fibrin. The immediate result may be that the aneurysm becomes hard and shrunken, pulsation diminishing or ceasing. The operation may be repeated after a few days' rest. It has been objected that the coagula formed by this proceeding are likely to be produced in the central portion of the sac and to be soft and apt to melt away rather than firm, laminated and adherent to the parietes. In some of the cases, however—as in one recorded by Dr. Ord—firm, laminated clot, at least half an inch thick, has been found adherent to the walls of the sac, the densest portion being at the spot where the negative electrode had been introduced.

There are, nevertheless, many dangers attending the operation. In a case, under the care of Mr. Holmes, of aortic and innominate aneurysm which came close to the surface and was accompanied by great dyspnœa, Dr. Althaus practised the electrolytic treatment. Great pain was experienced during the operation and arterial blood spurted freely from the punctures. Diffuse cellulitis followed and the patient died in a few days. A like result occurred in a patient under my own care with an aneurysm of the ascending aorta pointing near the surface. Electrolysis was done with great care by Dr. de

Watteville, but cellulitis followed and the patient succumbed in a short time. It was my conviction that the patient's suffering was increased by the operation and that the policy of non-interference would have been better.

I cannot doubt from the evidence of recorded cases that galvano-puncture is a better method of treatment than filipuncture for cases of aortic aneurysm in which simpler measures have proved ineffectual. It has seemed to me that the needles used for insertion into the sac should be silvered or gilded, and that it would be better to leave them *in situ* after the current has been allowed to cease. If the surface were dressed antiseptically their presence within the sac would scarcely act prejudicially—not more so than the wires in filipuncture—and if a second electrolysis became necessary there would be no necessity for puncturing the sac again. The forcible withdrawal of the needles just after the electrolysis may be an element of danger.

Mechanical Compression of the Aorta.—This method of treatment is applicable only in cases of aneurysm of the abdominal aorta. The object is to arrest the flow of blood so that coagulation may take place in the sac. The compression may be above or below the aneurysm, on the proximal or distal side in its relation to the heart.

In all cases it is necessary that the patient be under the influence of an anæsthetic.

In proximal compression a properly arranged tourniquet is to be applied to the abdominal aorta about four inches above the umbilicus, the compressing pad being screwed down until pulsation in the aneurysmal sac and in the femoral arteries can no longer be felt. The duration of such compression has been in recorded cases from half an hour to twelve hours. If after the *séance* pulsation is felt to return, the proceeding should be repeated at an interval of a few days. In a favorable case the pulsation (perhaps after a few hours or even days subsequently to the compression) will diminish or cease and no pulse may be felt in the femoral arteries; the aneurysmal tumor will become hard and in course of time may dwindle even to extinction. In a case under the care of Dr. William Murray, of Newcastle-on-Tyne, in 1864, thus treated pulsation in the aneurysm ceased a few hours after the second application of pressure, and the patient was able to earn his living by manual labor for six years when he died from the bursting of a more recently formed aneurysm above the former.

The post-mortem examination showed that the old aneurysm had been converted into a fibrous mass and the aorta above it occluded. Collateral circulation within the abdomen had been carried on between branches of the superior and inferior mesenteric arteries, between the

upper lumbar and ilio-lumbar arteries, and between the lower lumbar arteries and the circumflex iliac. The superior mesenteric artery was dilated so that it was nearly the size of the aorta itself, while the inferior mesenteric had become dwindled to such degree that its calibre was much less than that of its branches. Collateral circulation outside the abdomen had been established between the internal mammary artery and the deep epigastric; between the intercostals and the epigastrics, superficial and deep; between the hepatic artery and a branch of the epigastric, and between the lower intercostals and the circumflex iliac. There are at least five recorded cases in which there has been cure of abdominal aneurysm after mechanical compression of the aorta on the side nearest to the heart (Philipson, *British Medical Journal*, March 9th, 1878, p. 331; Pringle, Medico-Chirurgical Transactions, 1887, p. 271; Lunn and Benham, *ibidem*, 1885, p. 191).

Unfortunately the large majority of cases of abdominal aneurysm cannot be treated by proximal compression of the aorta because seventy-five per cent. (133 out of 177 cases according to the late Dr. Sibson) arise from the aorta immediately below the diaphragm. In aneurysms which come from the abdominal aorta about the level of the cœliac axis mechanical compression of the vessel above the aneurysm is impracticable.

The point should be determined whether firm digital compression above the site of the aneurysmal tumor arrests pulsation in the sac, and if not, whether pressure on the vessel below the tumor, about the level of the umbilicus, sensibly diminishes the pulsation. In these latter distal compression has been practised. In recorded cases the pad of the tourniquet has been made to compress the vessel just above the umbilicus and a little to the left of the middle line. The records in five cases in which this method of distal pressure has been adopted are far from encouraging. It is true that in a case recorded by Lunn and Benham the sac became nearly filled with clot, but the patient died eleven days after the operation from embolism of the superior mesenteric artery and consequent gangrene of the intestine. In another case of Mr. Barwell's the compression was continued for six days without any benefit. The other dangers of the operation have been proved to be obstruction of the bowels, peritonitis, and softening of the pancreas.

My conclusion is that distal compression of the abdominal aorta in cases of abdominal aneurysm is never to be recommended. In the cases in which it is possible to adopt the method of proximal compression this should be attempted but with great care, for there are risks of peritonitis from the mechanical violence done to the abdomi-

nal contents, and of plugging of any of the arteries proceeding from the aorta with consequent gangrene of the organs within the abdomen or of the lower extremities.

Digital and mechanical methods of compression of the carotid and subclavian arteries have been practised in cases of supposed innominate aneurysm, but in reality aneurysm of the thoracic aorta. An interesting case has been recorded by Dr. J. McFadden Gaston, of Atlanta, Ga. (*International Clinics*, 1893, p. 115). No material improvement followed and a subsequent operation of ligature of the common carotid artery was performed. During the progress of this operation the internal jugular vein was found much enlarged and its coats thickened and agglutinated to the artery beneath. This is a sufficient commentary on the dangers of mechanical compression. Sudden death occurred immediately on the closure by ligature of the common carotid artery. At the post-mortem examination it was found that the aneurysm which seemed to be of the innominate artery, was really one of two saccular aneurysms arising from an immense dilatation of the entire arch of the aorta.

Injection of drugs into the sac of the aneurysm in order to cause coagulation is fraught with danger and cannot be recommended. Perchloride of iron has been used for this purpose. Fibrin ferment (lecithin) has been suggested but has not come into the sphere of practical therapeutics.

Anæsthesia by Inhalation.—For some of the operative procedures just described the administration of an anæsthetic is absolutely necessary. I am convinced that such administration can be a valuable therapeutic measure in many cases previously to, and perhaps in substitution of, surgical interference.

The administration of an anæsthetic is especially valuable in those cases of aortic aneurysm, thoracic or abdominal, in which severe pain (continuous or paroxysmal) is a feature. It may be employed when antipyrin and phenacetin are inefficacious, when subcutaneous injections of morphine are not well borne or cease to relieve the pain except the dose be increased, when distressing pulsation and discomfort prevent sleep, and—previously to any surgical interference—when there is alarming increase in the proportions of the aneurysm. The effect of the prolonged sleep produced by an anæsthetic is far more potent for good than that of the other measures for the relief of pain. It affords time for coagulation within the sac, and when once it is shown that the inhalation can be managed with comfort, the administration may be repeated daily for several hours until a favorable change has come.

Chloroform is in the first instance to be preferred to ether because

the anæsthesia is brought about with much less muscular agitation on the part of the patient. It must be administered, however, with great care. No one is more convinced than myself that the reckless method of administering chloroform, as now too often practised, is fraught with danger. A handkerchief sprinkled or soaked with pure chloroform can give to a patient an atmosphere containing from ten to thirty per cent. of chloroform vapor, whereas precise observations tell us that an atmosphere of one or two per cent. is sufficient to produce and maintain anæsthesia. I advise that either a Junker's inhaler, as modified by Krohne and Sesemann, be employed or else that the pure choroform, used for sprinkling on the light cambric material made to cover the nose and mouth, be diluted with an equal volume of pure alcohol—as nearly absolute as can be procured. The alcohol effectually restrains the volatility of the chloroform so that a dangerous percentage is not given off. After the anæsthesia has been commenced by chloroform it can be continued by ether, and in some cases it may be found better to substitute pure ether for chloroform.

In my opinion the production and maintenance for some hours of anæsthesia in this manner—the proceeding to be repeated for several days—should precede the operative measures described. Under the anæsthetic sleep (in cases of thoracic as well as abdominal aneurysm) a diagnosis can be better made, and, as I have said, the measure can be looked on as not only palliative but curative.

Other Diseases of the Aorta.

Congenital malformations of the aorta and its valves are without the scope of this article. It is to be remembered that the vessel may be constricted just beyond the point where it is joined by the Ductus Arteriosus Botalli, the affection being known as coarctation of the thoracic aorta; it is compatible with even a considerable length of life (twenty-one to fifty years, Peacock). The most characteristic sign in such cases is enlargement of the arteries arising from the arch of the aorta. There may be such enlargement and pulsation of the intercostal and epigastric arteries as to simulate aneurysm of the transverse portion of the arch of the aorta or of the innominate artery.

The aorta may be abnormally narrow throughout its whole course. Virchow pointed out that this was the case in some instances of persistent chlorosis in young women. This question will be further considered in relation with narrowing of the pulmonary artery. The aorta is of less calibre than normal in the subjects of disease of the mitral valve arising in infancy and childhood, which causes a small volume of blood to be projected into the great artery during the years in which the development and growth of the heart are proceeding.

Diseases of the Pulmonary Artery.

Acute and Subacute Endarteritis.

Although the instances are very rare, there can be no doubt that inflammations do occur in the coats of the pulmonary artery and present appearances resembling those in analogous affections of the aorta.

Morbid Anatomy.—The lesions have been in some cases found associated with those of ulcerative endocarditis. Ulcerative endocarditis may attack the semilunar valves of the pulmonary artery, and patches showing inflammatory changes may extend upward in the lining membrane of the vessel. In association with such acute pulmonic endarteritis there may be constrictions of the branches of the pulmonary artery, resembling those found in the branches proceeding from the aorta in some instances of acute aortitis. In a case recorded by Fenger—that of a young man aged nineteen—there were found many vegetations on the semilunar valves of the pulmonary artery and a large number of others at the level of the bifurcation of the artery, which were prolonged into each of the branches. The comparatively slow evolution of the changes in the more distal portions of the artery and in its branches would explain the stenosis observed in some cases.

An interesting case has been recently recorded by Shingleton Smith in which it would appear that the lesions might have been the consequence of influenza. The left wall of the pulmonary artery in a direction upward from the left semilunar cusp was found to be inflamed and softened, a mass of fungating vegetations adhering to it. The trunk of the vessel was somewhat dilated, especially at its bifurcation, but just beyond this point there was a sudden contraction of the right branch leaving an opening not larger than one which a No. 4 catheter would traverse. Beyond the stenosed portion the artery was of the usual size, excepting at its origin from the main artery where there was a saccular dilatation. In the dilated trunk of the vessel were numerous detached masses of firm consistence and of pale yellow color or covered with a thin layer of recent blood-clot. They had evidently been churned by the eddying blood; they were too large to pass through the constricted right branch, but there seemed to be no obstacle to their passage through the left. Sections of these masses showed micrococci in great numbers, some of these being streptococci. There were evidences of numerous old infarcts in both lungs (*Medical Press and Circular*, August 29th, 1894).

In a case recorded by O. Weber the lesions within the pulmonary artery were just like those of aortitis, and were found in a woman who presented undoubted signs of syphilitic affections of the spinal cord and of the liver. There were in the right branch of the pulmonary artery, patchy elevations of yellow color and soft but yet covered by

the smooth internal coat. They projected into the vessel, obstructing its calibre so that its opening appeared as a mere slit. The external coat was supple but much thickened. Infarcts were found in both lungs, especially the right, and death took place from hæmoptysis (*Schmidt's Jahrbücher*, Bd. 123, p. 174).

Cases of stenosis of the right branch of the pulmonary artery have been recorded by Willigk and Vimont, and one instance in which both branches were thus affected is mentioned by Constantin Paul as observed by Tommasi Crudeli.

Causes and Course.—From the extant evidence it would seem that acute and subacute pulmonic endarteritis is in the largest number of cases due to the extension of the lesions of septic endocarditis and that the inflammation may be due to syphilis and probably also to influenza.

In Dr. Shingleton Smith's case, that of a lady aged 28, it was thought that there might have been some congenital affection, but the evidence, both clinical and pathological, makes this very doubtful. At the time of an attack of influenza a loud basic murmur was heard, and about twelve months afterward this murmur was found to be double with a continuous venous-like hum heard at the second left interspace. There were on many occasions attacks of dyspepsia, but the general condition was not indicative of any active disease for nearly three years subsequently to the last observation, when an attack of pneumonia occurred. There were repeated attacks of pneumonia during the succeeding five months, the basic murmur became very loud and rough in the area of the pulmonary artery, and a vibratile thrill was noticed at the third left intercostal space close to the sternum. The pyrexia, sweatings, and evidences of repeated pneumonic consolidations left little doubt that there were recurring septic embolisms of the lung, the sources of which were shown at the post-mortem examination to be the vegetations of endocarditis of the valves of the pulmonary artery and those derived from the diseased area, already described, of the pulmonary artery itself.

It is an interesting question whether in this case there were not repeated attacks of pulmonic endarteritis. It would seem probable that the most ancient lesion was the stenosis of the right branch of the vessel. Congenital lesions were conspicuous by their absence; the evidence of any cardiac lesion until the attack of influenza was very vague, but during this attack and subsequently, though there were long intervals of fair health, there is good evidence that the pulmonary artery became diseased, some of the lesions being chronic, others acute.

It appears to me that the case is analogous to cases observed by myself in which inflammations of the aorta were manifested after influenza. I have mentioned one in which death took place suddenly after signs which I considered to indicate acute aortitis. In another, that of a lady aged sixty-one, in whom influenzal toxæmia was proba-

ble but not certain, there were repeated attacks during three years, when symptoms of aortitis were manifested, and a diastolic murmur at the base indicated aortic incompetence. Lesions of chronic aortitis were shown at the post-mortem examination.

Chronic Endarteritis and Atheroma.

The signs of chronic inflammation and atheromatous changes are observed in the pulmonary artery much more rarely than in the aorta. From the tables of Rokitansky, Lobstein, and Huchard it would appear that the vessel is almost less liable to be attacked by atheroma than any other large artery in the body. Exceptionally, however, without doubt, it presents the lesions of atheroma.

Morbid Anatomy.—The appearances are identical with those seen in chronic inflammation and atheroma of the aorta. The areas of local morbid change show themselves at first as whitish or yellowish patches over which the smooth internal coat of the vessel extends; afterward, the morbid changes continuing, the external coat ulcerates and disappears through necrosis, and eventually there is incrustation with calcareous material. The patches are seen chiefly either in the neighborhood of the semilunar valves or at the level of the bifurcation of the artery. The trunk of the vessel becomes dilated in a similar manner to that of the aorta under similar conditions. The lesions are accompanied by hypertrophy of the right ventricle. Romberg (*Deutsches Archiv für klinische Medicin*, XCVIII., 1891) has recorded a case in which there was extensive sclerosis throughout the pulmonary artery and its branches with consecutive hypertrophy of the right ventricle. The patient, a man aged twenty-four, had suffered from severe dyspnœa, increased by exertion, with much blueness of the surface and of the mucous membranes, the first symptoms occurring more than fifteen months before his death. No cause could be traced for the affection and there was no congenital abnormality. Dr. James Barr, of Liverpool, has observed that in a majority of instances when atheroma of the pulmonary artery is observed post mortem this is in association with obstructive disease of the lungs or mitral-valve diseases, especially mitral stenosis. Constantin Paul has described a case in which the semilunar valves of the pulmonary artery were thickened, indurated, and cohering so that the opening into the vessel only admitted the little finger. Beyond the valves the trunk of the artery was dilated and its walls thinner than normal. No congenital anomaly of the heart or vessels was discovered. Many other such cases have been recorded, and in some of these it seems probable that a rheumatic form of endocarditis involved the valves of the pul-

monary artery. Such instances, however, are extremely rare; in a larger number of cases the endocarditis has been of the septic or ulcerative form, attended by exuberant vegetations blocking the valvular orifice, or by thickening and fusing of the cusps when the disease process has been slower.

In the majority of instances hypertrophy of the right ventricle has been observed. Almost invariably there has been found tubercular disease of the lung in cases where chronic obstructive disease has existed at the orifice of the pulmonary artery or in any part of the course of the vessel.

In the great majority of cases in which the valves of the pulmonary artery have been thickened the sclerosis has attacked congenitally abnormal valves. I have observed a case in which the valve was formed of two cusps only, and stenosis of the orifice was produced by the thickening of these and their fusion into a cone. The patient died at the age of twenty-one. There was no other congenital anomaly. In most cases a congenital malformation of the pulmonary artery is accompanied by an opening in the septum between the ventricles or by some other imperfection of development.

Diagnosis.—In acquired disease of the pulmonary artery the chief points to be relied upon for the diagnosis are (1) a systolic murmur heard superficially over the situation of the pulmonary artery, its maximum of intensity being the second left intercostal space near the sternum; very rarely a diastolic murmur. The second sound is in some cases louder than the normal, in others it is faint or inaudible. The systolic murmur may be very prolonged and may be followed by a prediastolic murmur ending abruptly with the second sound; (2) a visible pulsation of the dilated trunk of the vessel; (3) a strong pulsation felt over the right ventricle with signs of hypertrophy and dilatation of the right cavities, but not of the left; with this evidence of a strongly working right ventricle is found a radial pulse of small volume; (4) a thrill felt over the situation of the pulmonary artery with the ventricular systole; (5) the coexistence, or the sequence in a chronic case, of the signs of tubercular disease of the lungs.

There may be considerable difficulty in determining whether the pulmonary stenosis is *congenital* or acquired. In most cases inquiry will elicit the fact, in cases of congenital anomaly, that there has been some respiratory trouble or tendency to lividity, especially on exertion, from the period of infancy or early childhood. On the other hand, in acquired obstruction there may be a history of comparatively recent development of symptoms. In a patient past the prime of life there may be observed such evidence of degeneration of

the systemic arteries as would suggest the probability of atheromatous change in the pulmonary artery. The question whether there are any signs of septic endocarditis must be carefully considered.

It must be remembered that a systolic murmur over the site of the pulmonary artery is very frequent in *chlorosis* and *anæmia;* the murmur passes away as the patient improves in health. The probabilities in a given case are that a systolic murmur heard under the circumstances named is *not* due to structural disease in the valves or in the vessel. I have observed, however, the very loud murmur of pulmonary stenosis with extreme anæmia and no cyanosis in a child who died after having manifested progessive tubercular disease, and after death I found no local stenosis but simply a very small pulmonary artery. The vessel and its valves were minute but perfect. I have also observed a few cases in which in very anæmic young girls there has been a systolic murmur over the pulmonary artery with signs that I could not differentiate from organic stenosis. I consider that in these cases the pulmonary artery is abnormally small, and it is probable that the contracted aorta described by Virchow in such cases is but the sequel of a primarily deficiently developed pulmonary artery).

The danger of tuberculosis arising in such patients is great, but means directed to physically expand the chest and cause increased inflation of the lungs are sometimes effectual in improving the conditions and ultimately causing the disappearance of the murmur and the other physical signs indicating abnormal conditions of blood-current within the pulmonary artery.

Evidence as to Insufficiency of the Pulmonary Semilunar Valves.

A diastolic murmur generated at the orifice of the pulmonary artery is very rare. It may be heard over the second left intercostal space (or have its maximum in this situation) and may be audible down the sternum to the apex of the right ventricle. It may be due to (*a*) congenital malformation of the valves, with subsequent morbid changes; (*b*) ulcerative (septic) endocarditis, which may be protopathic or secondary, the morbid process in the latter case involving previously diseased structures; (*c*) aneurysmal dilatations of portions of the vessel.

In a case of Dr. Ormerod's, a man aged twenty-eight, a systolic murmur over the pulmonary artery became diastolic after the lapse of a year; on post-mortem examination the pulmonary valves were found agglutinated to form a thick cartilaginous ring, the orifice being of the size of a crow quill.

In a case recorded by Dr. Wilks, of a man aged thirty-one, there

was a double (systolic and diastolic) murmur over the pulmonary artery. The post-mortem showed that two segments of the semilunar valves had disappeared, the remaining one being normal. In other recorded cases there have been the signs of ulcerative endocarditis. Duroziez holds that insufficiency of the valves causing regurgitation from the pulmonary artery is not necessarily attended by a diastolic murmur. He cites two cases observed by Frerichs in which such a murmur was absent; in one of these the sigmoid valves were transformed into a diaphragm pierced by a triangular aperture, and there must of necessity have been regurgitation.

It has been thought, on the other hand, that there might be the diastolic murmur of regurgitation from the pulmonary artery when the vessel itself was not diseased but dilated from overdistention. Graham Steell has thus explained a diastolic murmur heard in cases of mitral stenosis. I have observed this murmur, but I am inclined to consider it an early diastolic murmur generated at the mitral orifice, but conducted toward the septum ventriculorum and thence to the sternum near the third intercostal space.

Aneurysm of the Pulmonary Artery.

This condition is very rare. Crisp collected from English records only 2 cases to 175 of aneurysm of the thoracic aorta, and found but 2 specimens in London museums to 207 of thoracic aneurism.

A case of general dilatation of the pulmonary artery with small saccular aneurysms above the semilunar valves has been recorded by Tommasi Crudeli (*Rivista Clinica*, VII., 2, p. 37, 1868), in which a murmur replaced the second sound, and was most marked over the right ventricle. A harsh whistling murmur was said to have been heard between the two heart sounds. The patient died suddenly while at breakfast. At the autopsy the trunk of the pulmonary artery was found dilated, and above the semilunar valves were two little diverticula, the walls of these pouches being only half a millimetre in thickness while the wall of the dilated pulmonary artery had a thickness of from one to two millimetres.

In a case recorded by Dowse (Transactions of the Pathological Society of London, 1866) a loud systolic murmur and thrill were manifested over the situation of the pulmonary artery. Death ensued from pericarditis. At the post-mortem examination a globular aneurysm, of the size of a hen's egg, filled with firm clot, was found at the origin of the pulmonary artery; the sac had ruptured into the pericardium. Vegetations were found on the semilunar valves; there was dilatation of the right ventricle, inducing tricuspid regurgitation.

In a case of Sir Dyce Duckworth's, at the post-mortem examination the valves of the pulmonary artery were found to be two in number, one large and one small, both with thickened edges; between the two was an aneurysmal pouch which would have held a large hazelnut. The heart was very large, the right cavities being especially dilated. The aortic valves were normal. It was considered probable that this was a case of congenital anomaly of the pulmonary valves, the consecutive heart changes occurring only in middle life. The patient was a man aged forty-nine, invalided from the police on account of cough and dyspnœa.

A case has been described by Dr. Bruen of a young girl, the subject of syphilis, who, at the age of twenty, presented signs of heart disease, a pulsating tumor being manifested on the left side of the sternum, between the second and fourth ribs, near the sternal border. The patient was under observation for three and one-half years. At the post-mortem examination two of the semilunar valves of the pulmonary artery were found to be nearly destroyed, and the third, much thickened, projected as a leaf-like fold into the mouth of the vessel. The pulmonary artery was dilated to twice its usual size. There were undoubted signs of congenital malformation of the heart. I consider that in this case, as in some of the others, septic endocarditis attacked the malformed structures.

Aneurysms of Branches of the Pulmonary Artery.

These are by no means rare in the subjects of tubercular disease of the lungs. The observations as to the infrequency of aneurysms of the pulmonary artery, therefore, do not apply to saccular dilatations of the branches. The rupture of these is often the cause of the profuse or repeated hemorrhages which induce death. A saccular dilatation is seen on post-mortem examination to spring from the wall of the artery, and to a rent in this the escape of blood can be traced. The aneurysms may vary from the size of a pea or hempseed to that of a small orange, the larger springing from the primary divisions of the main branch distributed to the lung. The aneurysm may almost completely fill a cavity in the lung. The cause of such aneurysms is probably peri-arteritis and endarteritis produced by the irritation of the bacilli and their toxins, the diseased wall of the vessel bulging into a cavity where it is unsupported by lung tissue. The aneurysms rarely, if ever, appear on those branches of the artery which are often found to traverse a vomica, but are found in those which exist in the thickness of the walls of the cavity. The branches of the artery in the diseased tissue around the cavity may be eroded without pre-

senting aneurysmal dilatations. Pyæmia, abscess, or gangrene of the lung may also induce such ulceration.

We may, I think, conclude that general dilatation of the pulmonary artery may occur in any case in which, owing to a narrowing at the valvular orifice, there is retention of blood in the portion of the vessel above the valves, or in cases where there is obstruction in the primary branches. The condition is analogous to a fusiform dilatation of the aorta. In an extreme case the vessel was so dilated that its circumference measured six and one-eighth inches, the normal average being three and one-half inches. Saccular dilatations of the trunk of the vessel are probably invariably due to septic influences or the mechanical irritation of vegetations. In all such cases infective emboli may be conveyed to the lung and set up pneumonia or abscess in the pulmonary tissue. Saccular dilatations of the branches are almost always due to the changes of pulmonary tuberculosis.

Lardaceous degeneration has been said to affect branches of the pulmonary artery in exceptional cases.

Diseases of the Systemic Arteries.

Acute and Subacute Arteritis.

It is rarely that an acute or subacute inflammatory change in the tissues of the arteries of the general arterial system can be traced. As in aortitis acute changes are sometimes found in the subjects of chronic disease, so, though the observations are still more rare, may the conjunction be found, in some instances, of acute and chronic systemic arteritis.

An interesting case has been recorded by Lancereaux[5]: A man aged 60 developed signs of aortic regurgitation with those of failing heart and pulmonary apoplexy. The left radial pulse was sudden and bounding, a typical "Corrigan pulse," while the right was barely perceptible. There was improvement in the symptoms for a time, but about six months after the first observation the patient died after having manifested profound cachexia, general œdema, and attacks of intense precordial oppression. At the post-mortem examination the aortic valves were found to be thickened and distended and the orifice contracted. Above the valves the aorta presented the signs of acute (or subacute), together with chronic, aortitis, calcareous degeneration being manifested in some parts. The orifices of the coronary arteries as well as those of most of the arteries which arise from the aorta were constricted. The right subclavian artery was so narrowed that it scarcely allowed the passage of a pin's head. In another case, recorded by the same author, of a woman aged 58 suddenly stricken with apoplexy, the post-mortem examination showed

a like concurrence of acute and chronic aortitis. Some of the elevations of the endarterium were soft, elastic, opaline or yellowish—others firm or encrusted with calcareous material. The changes were found throughout the aorta, thoracic and abdominal, except the first portion of the vessel. Almost all the branches springing from the aorta presented the signs of arteritis; some showed dilatations, others were notably contracted. The cœliac axis, the renal arteries, and the iliac arteries were thus markedly affected. The arteries of the base of the brain were the seat of opaline plaques slightly prominent in the inner coat. There was abundant intra-cranial hemorrhage. Lancereaux states that the whole arterial system was affected.

These cases and the few records that are available show that in conjunction with acute and subacute aortitis there may be a change in the coats of the arteries springing from the aorta whereby these latter are contracted in a remarkable degree (*constricting arteritis*). We have seen that a like change, under similar conditions, may take place in the branches of the pulmonary artery.

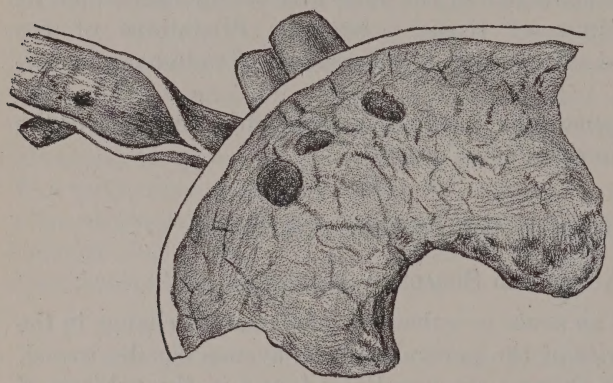


Fig. 35.—Interior of a Portion of the Aorta showing Chronic Arteritis with Constrictions of the Orifices of the Great Vessels. The innominate artery is seen to be contracted and rendered oval for a short part of its course, and the right subclavian artery is extremely small and almost impervious. (After Lancereaux.)

There is no evidence to show that these arterial inflammations, attended by contraction of the walls of the vessels, give rise to any special symptoms. It is possible that such changes in some of the larger arteries may explain the signs in some of the rare cases in which a very loud systolic murmur has been heard over the arteries. The loud sound was heard in a case recorded by H. Roger and Chairou over the secondary and tertiary arterial branches in all regions of the body accessible to the stethoscope. It was so intense as to be audible at a distance from the body, and rendered sleep almost impossible to the unfortunate subject of the malady (Barth and Roger: "Traité Pratique d'Auscultation," Paris, 1887, p. 528).

Acute local arteritis may occur in the case of an artery exposed in a wound to the influences of ulceration or suppuration, and where an embolus (septic or otherwise) has lodged against a portion of its

lining membrane and has thus set up an irritation. In the latter case it produces an aneurysmal dilatation as will be subsequently considered.

Arteritis in Infectious Diseases.

Inflammation of the arteries has been said to occur in typhoid fever, variola, diphtheria, scarlet fever, puerperal fever, measles, and influenza. Huchard contended that the morbid changes in the muscle of the heart, which have been abundantly demonstrated in many of these infectious diseases, were really due to endarteritis of branches of the coronary arteries. The thickening of the lining membrane of the small arteries, attended by the accumulation of leucocytes and the formation of fibrinous plugs, thus caused an obstruction of the vessel in whole or in part; hemorrhagic infarctions resulted. There were therefore two causes in operation to produce the myocarditis, (1) inflammation; (2) obstruction of the nutrient blood-stream. Brouardel also described endarteritis as accompanying variola, and Landouzy and Siredey studied these in typhoid fever and scarlet fever as well as small-pox.

These authors described the changes as involving all the tunics of the arteries at once, the finer arterioles being in greatest degree affected. The alterations in the muscular fibrillæ and the interstitial fibrous tissue are consequent upon the endarteritis; the muscular elements become atrophied and degenerated whilst the fibrous stroma proliferates. The process of degeneration is analogous to that which occurs in cirrhosis of the liver. Martin has found in diphtheria an extreme thickening of the internal coat of the arterioles not only in the lungs attacked with broncho-pneumonia, but also in the heart, and often in the kidneys. The evidence derived from a considerable number of observations tends to show that in many of the examples of acute arteritis occurring in infectious diseases the primary lesion is in the minute arteries of the wall of the vessel—the vasa vasorum. There is thus a strict analogy with the pathogeny of aortitis, acute and chronic, which we have already considered. Louis Thérèse (Étude Anatomo-pathologique et Expérimentale des Artérites Secondaires aux Maladies Infectieuses," Thèse de Paris, 1893) has traced the early changes beyond the arteries and the capillaries. He found in a case in which death had occurred from subacute aortitis that the patches of sclerous change in the middle coat of the aorta had in their axial portions a capillary vessel surrounded by exuded corpuscles. He found similar changes in small patches of acute aortitis observed in the case of a woman who died from puerperal septicæmia. The capillaries going to the diseased area (though endothelium and

innermost layers of the inner coat were intact) were surrounded by abundant round cells, and were obviously structurally modified. The conditions of disease could be traced from these capillaries, and the minute arteries in the external coat whence they sprung. Where the arterioles were healthy the capillaries showed no encircling masses of round cells. In some cases, however, the capillaries presented abnormal transudations when the tunics of the arteries themselves were healthy. The author concludes from his observations, both clinical and experimental, that the infectious diseases may be the initial causes of arteriosclerosis; that the first stage of the morbid change which is consecutive to these infectious diseases is a transudation of leucocytes outside the walls of the capillaries and into the interspaces of the connective tissue; that this transudation is an effect of certain toxins and that the transmigrated corpuscles undergo all the phases of inflammatory proliferation of fibre cells.

The conclusions of this observer generally accord with the results of my own observations, but I am disposed to think that the lesions of the internal layers of the intima are due to the indirect and not the direct influence of the leucocytes. The widening of the meshes of the fibres of the internal coat beneath the intact endothelium is a very early change. The fibres are separated, I believe, by a merely fluid exudation. The masses of leucocytes are much more distant from the endothelium. Agreeing that the morbid changes start from the capillaries and arterioles and are first manifested in the deeper layers of the internal coat, I think that a fluid exudation rapidly takes place which displaces the fibres of the layers subjacent to the endothelium, and in great degree causes the observed swellings of the internal coat.

It has been said that inflammation of the aorta as well as some of the systemic arteries has been observed in *acute tuberculosis*, but I do not know of a well-described case. In tubercular meningitis the arterioles of the brain and membranes may have their walls penetrated by bacilli and their lumen obstructed, and thrombosis may result. In acute miliary tuberculosis the walls of the arterioles of the lungs in the site of the nodules are thickened and infiltrated with cells and innumerable tubercular bacilli.*

Periarteritis.

Periarteritis is the term given to an inflammatory or proliferating affection involving the external coat of an artery, so that this shows

* See paper by Percy Kidd, M.D., on the Distribution of the Tubercle Bacilli in the Lesions of Phthisis. Medico-Chirurgical Transactions, vol. lxviii., 1885. Plate ii., Fig. 2.

diffuse or circumscribed thickenings, there being new formation of its connective tissue.

In the case of the cerebral arteries Lancereaux considers that syphilitic disease commences by periarteritis, the lymphoid tissue around the vessel being first attacked. Heubner, on the other hand, holds that the syphilitic lesion commences in the internal coat. Probably both observers are right, for the disease may commence in either way. Charcot and Bouchard adopted the view that the minute aneurysms, the bursting of which gave rise to intracranial hemorrhage, were the sequence of a morbid change in the perivascular sheaths. Lancereaux has adduced cases which show the probability that little circumscribed nodosities observed on the arteries of the base of the brain limited to the external coat may be associated with tuberculosis. Periarteritis has also been described as occurring in relation with the small arteries of the heart, the lungs, the kidneys, the liver, and most of the other viscera. It is, however, usually associated with endarteritis.

Chronic Arteritis—Atheroma of the Systemic Arteries.

(Endarteritis Deformans of Virchow.)

The morbid anatomy is essentially the same as that observed in atheroma of the aorta, and we may ascribe the series of changes—the accumulations of round cells, the subsequent fatty degenerations and calcareous incrustations, and the concomitant fibrous proliferations—to identical causes, viz., the entry into the fine branches of the nutrient artery or arteries of the affected area (the vasa vasorum) of minute intruding particles which either cause irritations of portions of the lining membrane of the affected larger arteries or else block completely the lumen of the latter.

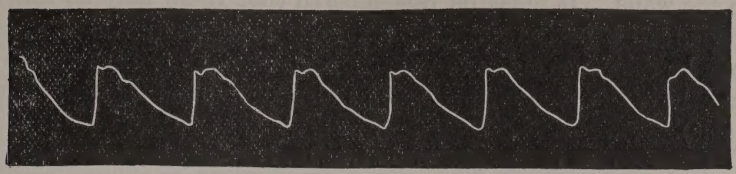
Incidence of Atheroma upon the Various Arteries.—Combining the observations of Rokitansky, Lobstein, and Huchard with my own, I think the following will fairly represent the relative liability of the various arteries of the body to the forms of atheromatous disease which are manifested after middle life. I would place the coronary arteries of the heart immediately after the aorta in regard to the liability to atheroma. I think its lesions have often remained undiscovered because the whole extent of the vessels has not been sufficiently examined. In this view I agree generally with Huchard. The systemic arteries in regard to their proneness to atheromatous change are in the following order: 1, the splenic and renal arteries; 2, the external iliac, femoral, and popliteal arteries; 3, the temporal, subclavian, carotid, and brachial arteries; 4, the arteries of the base of

the brain; and 5, the arteries of the stomach, liver, mesentery, uterus, and those of the thoracic and abdominal walls.

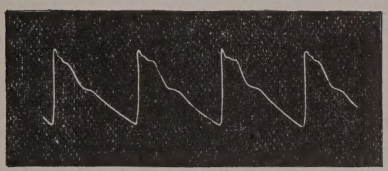
Clinical Evidence of Atheroma of the Arteries.—The arteries, the observation of which gives the most important evidence of atheroma, are the radials, the brachials, the temporals, and the carotids.

In the examination for the detection of atheroma, the *radial pulse* should be first felt in the usual way. The tips of the fingers of the right hand should exercise varying pressures upon the artery and should glide from side to side over the vessel. Then the observer should lay one finger lengthwise over the pulsating artery and push up the skin so that the characters of the coats in all its available length for exploration may be investigated. The vessel may be felt to be tortuous, presenting abnormal curvings and may roll under the finger as a hard cord; patches of irregular rigidity or of calcareous deposit may be detected within its walls; or the whole vessel may be hard, having a thick, leathery, or inelastic character; or it may be converted into a hard, irregular, calcareous tube which may feel somewhat like a string of beads under the finger. Dr. William Ewart observes that it is unusual for calcification to proceed evenly along any considerable length of the vessel; between the calcareous portions the artery preserves some pliability. It is very rare for it to be felt as a tube like the stem of a clay pipe, yet the calcareous patches may be set very close together rendering the artery extremely rigid. It is undesirable to exercise very considerable force in compressing such an artery. It is to be remembered that the patchy irregularities of the arterial wall are the signs characteristic of atheroma. The artery may be very firm and incompressible and yet not atheromatous. Arteriosclerosis, that we shall hereafter consider, is not atheroma, though the two conditions often coexist; but atheroma may be manifested without arteriosclerosis.

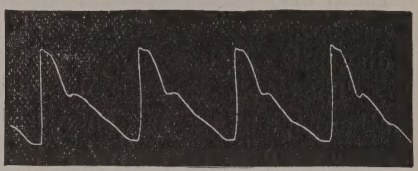
The *brachial artery* should be thus examined: The right arm as well as the right side of the chest should be uncovered, the patient should turn his face slightly to the left and the arm should be semiflexed at the elbow. If the vessel be atheromatous it will be seen that there is considerable side-to-side movement with each pulsation. If the arm be extended and lifted the movement greatly diminishes. It is well known that a very great displacement of the artery with bounding and throbbing pulsation is seen in cases of aortic regurgitation (the Corrigan pulse). Then the elevation of the arm increases the suddenness and visibility. The extension and elevation of the arm, therefore, serve to differentiate between the two conditions, and the diagnosis of atheroma may be confirmed by the examination of the artery by the fingers which may detect local patches of hardness



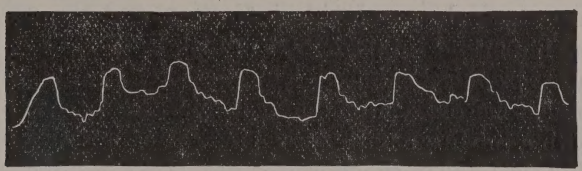
1. Senile Pulse. Patient aged 72 years; arteries very rigid; considerable hypertrophy of the left ventricle. (Finlayson.)



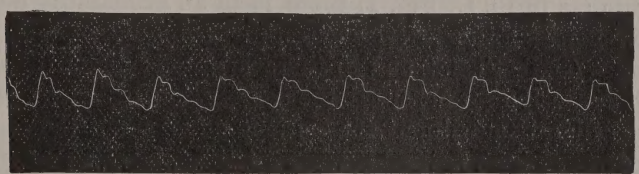
2. Sphygmogram in Atheroma. (Sir B. W. Foster.)



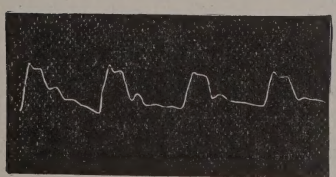
3. Atheromatous Artery. Pressure six ounces. (Frederick Taylor.)



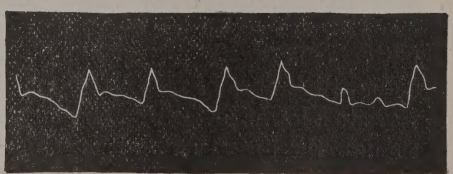
4. Senile Pulse; sudden and full, the trace resembling more or less that of the left ventricle. (Sir W. Broadbent.)



5. Pulse in a Man, Aged 49 Years, with Symptoms of Angina Pectoris; showing signs of atheroma in the brachial arteries, etc. (Author.)



6. Dicrotism in Atheroma in a Woman Aged 69 Years. Failing heart. (Author.)



7. Atheroma in a Man Aged 60 Years. Failing heart with irregularity. (Author.)

PLATE I.—SPHYGMOGRAMS ILLUSTRATING ARTERIAL ATHEROMA.

in the walls. It must be remembered that there may be atheromatous arteries in the subjects of aortic regurgitation and atheroma may be the cause of the valvular imperfection, and so the visible pulse may have the characters of the pulse of unfilled arteries (Corrigan pulse).

Observation of the *temporal arteries* gives important evidence. In ordinary health these arteries are scarcely visible. When they are tortuous and cord-like, hard and incompressible under the finger, there may be a general thickening of all the coats or atheroma in patches. Careful palpation will detect these patches and confirm the diagnosis of atheroma.

The *carotid pulse* may be more than ordinarily visible in cases of atheromatous disease, though the pulsations are not so strong as in cases of aortic regurgitation nor even as in cases of anæmia and in some neuroses. In atheroma, however, the pulse may be felt to be of unusual force and the finger may detect inequalities in the vessel. All these arteries should be explored. The finding of patches of atheroma in one may be a valuable indication, and the evidence will be still more important if thickenings are felt in more than one.

Sphygmographic Evidence.—The characteristics of the pulse-trace in atheromatous disease of the systemic arteries are a vertical upstroke with a broad summit-plateau indicating a prolonged first wave. It has been said that the dicrotic pulse is not manifested in old persons who have atheromatous arteries; this is an error. In the cases in which the dicrotic wave and notch are not manifested in the sphygmogram (as in Pl. I., Fig. 1) there is in all probability a concurring arteriosclerosis, the arteries generally being thickened. In many instances (as in Figs. 2 and 3) there is a well-marked dicrotic wave. I have scarcely ever observed the conjunction of broad summit and marked dicrotism without the patient manifesting the signs of failing heart. I have noticed it when there have been concurrent signs of hypertrophy and dilatation of the heart with atheromatous vessels. I have seen it in cases of alcoholism when degeneration of the heart might be suspected, in some instances of rapid heart and irritable heart in which commencing dilatation was probable, and in certain patients manifesting hypochondriasis or melancholia. In some cases a slight increase of pressure upon the artery brings down the dicrotic wave to a lower level (Fig. 6) and may quite extinguish it. In others, though the broad summit is still shown, the diastolic portion of the tracing is broken by many serrations, the rigid artery being very imperfectly filled with blood (Fig. 4). In another form the summit is sometimes pointed, at others slightly truncated, the feeble ventricle sending a varying but weak blood-wave into the rigid artery.

Ophthalmoscopic Evidence.—Rahlmann considers that an ophthalmoscopic examination of the retinal vessels will indicate atheroma of the cerebral arteries. The retinal arteries show a strangulation or constriction with diminution of their lumen; above and below the points of constriction are found streakings; at the constricted portions the blood-column is seen to be narrow and the vessel-wall either invisible or presenting a fusiform thickening. There may be over these spots some small hemorrhages without any inflammatory changes. The veins also may show analogous strangulations or sacciform dilatations.

Atheroma of the Arteries of the Brain.

The ordinary lesions of atheroma—the yellow patches of thickened tissue or the calcareous plates—may be observed in the arteries of the brain. The larger arteries of the base of the brain are the most frequent seats of these changes. In some cases one or two patches only are found, but usually the coats of many of the vessels are affected. The distribution of the disease in the arteries is sometimes symmetrical. In the majority of cases others of the systemic arteries are also atheromatous, but in some the disease is in the cerebral vessels only. The effect on a vessel may be (*a*) to narrow its lumen by the swelling of the internal coat, or (*b*) by the accumulation of the degenerated products or through a weakening of all the coats, to produce a dilatation. The arterial tube may be completely occluded. The small arteries springing from the vessel at its diseased portion may be obstructed though itself is pervious. Then

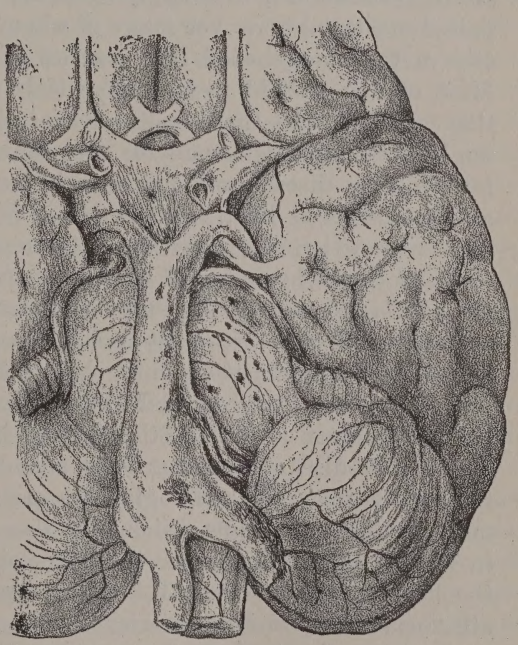


FIG. 36.—Chronic Endarteritis of the Basilar Artery, with Fusiform Aneurysmal Dilatations of the Trunk of the Vessel; occlusion by coagulation, and consecutive softening of the pons Varolii. (After Lancereaux.)

commonly the products of disease within the vessel act as foreign material and induce coagulation of the circulating blood—thrombosis. If the clot obstructs the middle cerebral artery or its branches, softening occurs in the great central ganglia; if the branches of the basilar are obstructed there ensues softening of the pons Varolii. In cases in which life is sufficiently prolonged, the clot within the vessel may become organized or may degenerate and even undergo calcareous transformation. The artery may dwindle and be converted into a fibrous cord.

Symptoms and Treatment of Chronic Arteritis (Atheroma).

In the senile form of systemic arteritis medicinal treatment is in many cases unnecessary and impossible. Instances must be known to many of us of vigorous people of both sexes, advanced in age, who are in possession of seemingly excellent physical health and unimpaired mental powers, yet many of whom have, as an almost necessity of their lengthened life, patches of disease in their arteries. Most of them would resent, and wisely resent, any suggestion that they should make their bodies receptacles for drugs, though they might listen to judicious advice concerning the means to be adopted for avoiding disease and for ministering to a healthy nutrition by a well-considered dietary. The advent of acute disease or some disorder of function may, however, bring under the notice of the physician a patient who is found on examination to present decisive evidence of atheroma of the systemic arteries. I would suggest that then the method of treatment should be considered after the observer has made an investigation which has resulted in placing the case in one of the following groups: I. The outline of the heart and the position of the apex are within the normal limits; the aorta shows no evidence of disease and the heart sounds are not heard to deviate from the normal. II. Though there are no marked physical abnormalities, there are signs which give rise to a suspicion of atheroma in the coronary arteries or there are signs of organic disease of the heart or its valves. III. There are symptoms which suggest atheroma of the cerebral arteries.

In Group I., signs of atheromatous disease are found in the systemic arteries. The apex beat of the heart is felt in its normal situation or slightly to the right of this; it is of ordinary force; it may not be detected because the aërated lung covers it, but plessimetric percussion shows that the heart is enlarged neither in its left nor in its right chambers.

We will assume that a patient presenting these signs has come

under our notice for symptoms of indigestion, for some disturbance of the action of the heart, or for a painful affection referred to the chest in the near neighborhood of the heart. Certain preliminary considerations come before us. It has been the teaching of some that the heart of aged persons is necessarily enlarged, that the heart is always hypertrophied in advanced life. This was a conclusion forced by the elaborate series of weighings of Bizot and confirmed by Clendinning and Peacock. The more extensive investigations of Robert Boyd, having their basis in 2,045 post-mortem examinations made at the St. Marylebone Infirmary, showed that the mean weight of the heart was at its maximum in the male between the ages of 70 and 80 and in the female between 50 and 70. Between the ages of 80 and 90 in the male and 70 to 90 in the female this maximum mean weight became notably reduced. Wilhelm Müller in 1883, though his observations were in a smaller number of subjects (433), carried his investigations out with such minute precision that his conclusions are of much value and serve to correct some erroneous deductions. His figures showed the relative liability to arteritis at the various ages. While in the subjects which he examined there were evidences of endarteritis in 32 per cent. of those between 31 and 40 years, the figures for those between 61 and 70 were 95 per cent., between 71 and 80 92 per cent., and between 81 and 90 100 per cent. He showed that the intrapericardial portion of the great arteries progressively increased in weight with age, no doubt concurrently with the liability to atheroma. The mean weight of the muscular mass of the heart was at its maximum between the ages of 61 and 70 in both male and female subjects—at later ages there was much reduction. It is incorrect, therefore, to say that the weight of the heart necessarily increases progressively with old age. No doubt with the tendency to atheroma there is usually a tendency to hypertrophy of the heart, but in those over 70 years of age it may be that the heart shrinks and wastes. As of the average so of the individual. I am sure that many observers have, like myself, witnessed cases of old people with well-marked atheroma of arteries whose hearts have presented no signs of increase above the normal size; in some they have seemed decidedly below the normal.

In such cases a careful regulation of the diet, which must not be too restricted, is of first importance. Alcohol must not be taken except with food; but the allowance may be rather larger than in younger subjects. If brandy or whiskey be taken, the maximum per day should be three ounces, and of this quantity one ounce should be taken beaten up with an egg and hot water or added to hot milk at

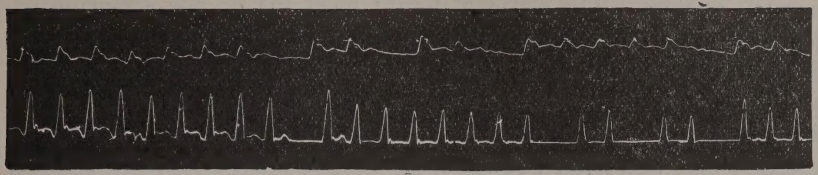
night, or night and morning, the remainder being taken well diluted with the meals. In those who object to alcohol, hot water or hot water with milk added should be taken with the meals. If claret or similar wine be taken, this should be in quantities not greater than two claret glasses with each meal, hot water being sipped therewith. The clothing should be especially light and warm, the underclothing woollen. There should be a fair amount of exercise in the fresh air—a locality being chosen, if possible, where, without extremes of temperature, there are warm sun and blue sky. Dyspepsia in such subjects is best treated by alkalies. Acids seldom agree. The following prescription may be cited as useful: ℞ Potassii bicarbonatis, sodii carbonatis, āā gr. x.; ammonii carbonatis, gr. iij.; bismuthi carbonatis, gr. x.; menthol, gr. ½; in a powder to be taken stirred in half a wineglassful of water three times a day after food. Vichy or Carlsbad water may be in some cases administered in addition or instead. "The senile heart," says Dr. G. W. Balfour, "is the gouty heart." The dyspepsia of the subjects of atheroma is a gouty dyspepsia, at any rate disturbances like those associated with gout are frequently produced. Any tendency to constipation should be rectified, but strong purgatives acting suddenly and powerfully are generally to be deprecated. The choice should be between cascara preparations, Gregory's powder, magnesia mixture, or the Hunyadi or Æsculap waters.

It is perhaps most important to advert to what not to do. I would generally deprecate the administration of the salts of iron and, as a working rule, digitalis and all forms of cardiac tonics. I have often known the senile subject of atheroma to be persuaded by advertisements or by the injudicious advice of friends to take a course of some of the iron preparations. The result is frequently disastrous. The old do not generally suffer from want of hæmoglobin; they get a sufficient daily supply from the meats and gravies of their ordinary diet. Lichtenstern found that the normal proportion of hæmoglobin in the blood was always increased after the age of sixty. If an aged subject with atheroma presents a marked anæmia, malignant disease should be suspected, or renal disease, or some affection attended with loss of blood. The iron salts tend to cause or to increase the muscular contraction of the arterial walls. Digitalis has a decided effect in contracting the arterioles. In atheroma, even if there be no association with the generally thickened and constricted arteries, the induction of a contraction whereby the arterial blood-pressure is raised is a harmful proceeding. Persistence with the treatment by alkalies with occasional additions, for a few days together, of the iodides will often do much good, while iron and digitalis do harm.

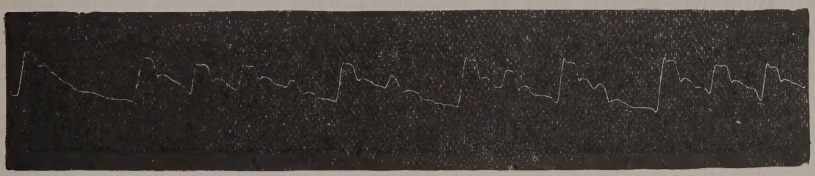
As a digestive and an appetizing tonic, quinine (gr. i. to gr. ij.) with dilute hydrobromic acid ♏ xx. (in some cases with the addition of liquor strychniæ ♏ v.), administered with a sufficiency of water three times a day, often does much good, and the hydrobromic acid does not fall under the general condemnation of acids. Floating on this mixture flavored with lemon, cod-liver oil in doses of two to four drachms may be administered in cases of failing nutrition. Where cod-liver oil is not well tolerated by the stomach, I have seen excellent results follow its administration as a nutritive enema. The plan I have long adopted is this: A wide-mouthed glass bottle with a capacity of about six ounces is procured. Into this is broken one egg; four tablespoonsful of hot milk and two tablespoonfuls of cod-liver oil are added, and all are shaken together to form an emulsion which is to be slowly administered per rectum—the supply tube of the india-rubber enema syringe is to be inserted through the wide mouth of the bottle so that the emulsion is administered from the bottle itself. The patient should remain very quietly recumbent afterwards so that, if possible, all may be absorbed. Such enema may be given night and morning, and more frequently if required. An advantage of this plan, besides that of being the most efficiently nutritive enema I know, is that it keeps the rectum free from all irritating matter, and, though it may be retained for hours, it prevents constipation. This method of treatment by no means precludes exercise. After quiescence for half an hour or one hour, the unabsorbed part of the enema is retained, the bowel being perfectly tolerant of it.

A patient, male or female, with atheromatous arteries may consult a physician for some *disturbance of the action of the heart.* There may be palpitations or protracted undue rapidity of the heart's pulsations, or irregularities of its action or abnormal slowness. Such symptoms are regarded with much dread by the patient. They appeal very strongly to his apprehension, and implant within him a not unreasonable fear that there is a condition of serious disease of the heart which may prove fatal in the near future. They are causes of frequent alarm and an abiding sense of insecurity. As to the real significance of these symptoms there are differences of opinion even among skilled observers. According to a physician who speaks with high authority, palpitations, rapid action, intermissions, and irregularities occurring in an old person always indicate debility of the muscle of the heart—a debility which, left to itself sooner or later tends to dilatation of the heart-chambers, with all the serious consequences which flow from this condition.[12] Some other physicians are more inclined to accept the view that a disturbance of the nervous mechanism of the heart has a considerable share in the pro-

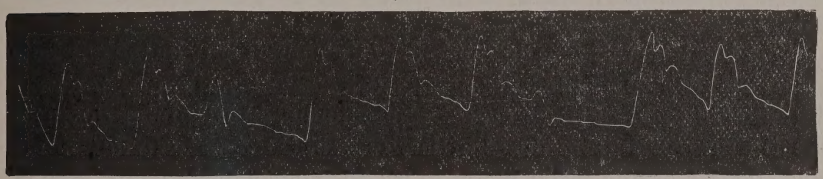
duction of the phenomena, but opinion is not formed as to the relative share of the nervous and muscular elements in the production of the phenomena. From the study of a considerable number of cases [13] I have been led to the conclusion that the disordered conditions of the cardiac rhythm, whether in the sense of abnormal rapidity (tachycardia), irregularity (arhythmia), or unusual retardation (bradycardia) are essentially due to disturbance of the nervous mechanism of the heart and cannot be taken as practical criteria of any affection of the myocardium. It is true that the chambers of the heart may suffer—in a minority of cases they become dilated—but this change is in the position of a consequence, not of a cause. The dangers of the symptoms consist partly in this dilatation and partly because the various disorders mentioned may be engrafted upon a heart already affected by organic disease. The causes of palpitations and of intermission and irregularity of the heart's action are often reflex, the most common being an error of diet. The omission from the daily dietary of tea in some cases, potatoes and sugar in others (Pl. II., Fig. 1), has served to abolish the distressing symptoms. In many there is a more persistent condition of disturbance in those portions of the nervous system—the roots of the vagus, the medulla oblongata, the cervical part of the spinal cord, the vagi and the sympathetic nerve-filaments and ganglia—which have to do with the normal cardiac reflex. In some cases the initiatory causes can be traced to a distinct shock, mental or physical—in these not infrequently the usual signs of Graves' disease are also concurrently induced. I have found extreme irregularity of the heart associated with a considerable number of cases of Graves' disease, though the rapid heart occurs in the majority. In other cases the instability of the portions of the nervous system mentioned can be traced as the effect of an acute disease, especially influenza (Pl. II., Fig. 5). The morbid affection of the nerve elements alone suffices to bring about the rapid heart, the irregular heart, or the slow heart for protracted periods; it also induces an "irritable weakness" whereby reflex causes—*e.g.*, such as bring about dyspepsia—provoke and increase the symptoms. Abnormal acceleration of the heart's action may occur at almost any age; it probably affects the old less frequently than young adults. At all ages dilatation of the heart may follow but does not precede the tachycardia unless due to pre-existing causes, and such dilatation is not observed in the majority of cases. A patient having atheromatous arteries who complains of palpitations, there being no evidence of coexistent heart disease, should be treated by (1) a careful regulation of the diet; (2) the administration of alkaline medicines on the lines already indicated; (3) the adoption of means to calm the



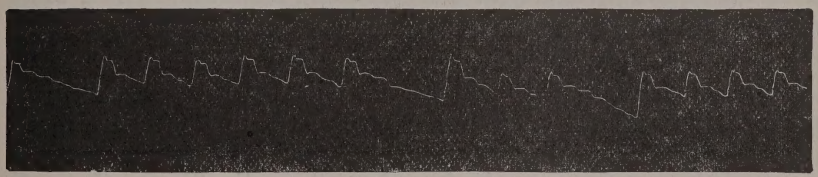
1. Man, Aged 64 Years. Irregularity and Intermittence. After omission of potatoes and sugar from the diet for fourteen days, no irregularity whatever was observed.



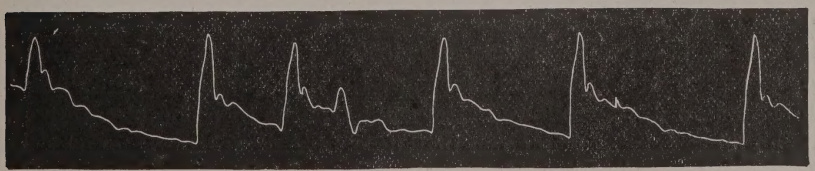
2. Man, Aged 71 Years, with Atheromatous Arteries. Irregularity observed for four years. Very little subjective discomfort.



3. Man, Aged 74 Years, Suffering with Tinnitus Aurium and Auditory Vertigo.



4. Woman, Aged 55 Years, Suffering with Tinnitus Aurium and Auditory Vertigo. Irregularity observed during a period of more than three years. No heart symptoms.



5. Man, Aged 62 Years, Gouty. Irregularity observed seven months after an attack of influenza. Patient unconscious of the irregularity.

PLATE II.—SPHYGMOGRAMS SHOWING IRREGULARITIES IN THE SENILE HEART IN CASES IN WHICH THERE WAS NO EVIDENCE OF HEART DISEASE AND LITTLE OR NO HEART DISCMFORT.

nervous system. The patient should have his or her attention directed away from the heart; it should be explained that it is the digestion that is chiefly at fault. Insomnia is a frequent association and must be treated. The bromides, especially the bromide of sodium in 20-grain doses, one dose taken in the evening and a second on going to bed, are often effectual. To supplement or reinforce these, chloralamid in 20 or 30 grain doses in cachets or dissolved by triturating with a little spirit, some syrup of orange or of lemon being subsequently added, may be employed with advantage and without fear of any ill consequences. Chloralose, in doses of from 2 to 8 grains administered in cachets, is said to be even better than chloralamid. In some cases morphine is a necessity, the less powerful hypnotics failing to produce sleep. From a quarter to half a grain of the acetate, hydrobromate, hydrochlorate, or meconate of morphine may be given at night, but not every night. Care must be taken lest it lock up the secretions (so an aperient should be administered in the morning), and lest, by too frequent employment, the morphine habit be engendered.

The treatment of the more persistently rapid heart in the subjects of arterial atheroma presents greater difficulties. I have never seen digitalis do good in these cases. Belladonna, in 10-minim doses of the tincture or $\frac{1}{2}$-grain doses of the extract three times a day, is in some degree useful, but it is apt to produce uncomfortable dryness of the throat and some other of its unpleasant effects. It should be given for certain periods with corresponding intervals. When there is much mental excitement the hydrobromate of hyoscine in doses of $\frac{1}{200}$ to $\frac{1}{100}$ grain, administered subcutaneously, produces wonderful calm, but the injection should not be repeated within twenty-four hours. Hyoscyamine ($\frac{1}{50}$ grain) may be used in like manner. It must be remembered that the heart-beats may be persistently, for long periods, over 100 per minute and yet the patient be free from all uncomfortable symptoms. In these cases the enforcement of absolute rest often does more harm than good. Some not irksome occupation is advisable. I have found in cases of tachycardia that the use of a weak continuous galvanic current from the nape of the neck to the region of the vagus in front of the neck for six to ten minutes three times a day is followed after some months by a notable decrease of the pulse rate, and in some instances by a complete restoration to the normal.

The heart may be very notably irregular in its action in gouty, atheromatous subjects, and yet the patient may be entirely unconscious of the irregularity. Intermission of the beats may be evidenced for years, whenever the physician has an opportunity of ob-

serving the case, and yet the patient may seem none the worse. I have found the most extreme forms of irregularity in some patients who were quite unconscious that there was anything wrong with the mechanism of their hearts. It is in my opinion of the greatest importance that the physician who observes such irregularity should refrain from giving the patient the least indication that any abnormal action exists. Once the patient's attention is directed to the tumult, he will be distressed by it and will be too frequently feeling his pulse, putting his hand over the præcordium, and attending expectantly for the sensation of throbbing. In the section of cases I am considering, I do not believe that the irregularity appreciably increases the gravity of the prognosis. The case is to be treated chiefly by dietetic rules and the general plan laid down for the management of cases of tachycardia. In some cases colchicum is of use. Dr. G. W. Balfour says: "Throughout the literature of cardiac disease there are recorded many cases of extreme and distressing irregularity of the heart at once relieved by a fit of the gout; all of these would no doubt have been cured just as well and as speedily by the use of colchicum."

I have found a very marked association of irregularity of the heart with morbid conditions of the naso-pharyngeal passages and with disturbances of hearing. I have found it in many cases of tinnitus aurium and auditory vertigo (Pl. II., Figs. 3 and 4), and have known complete cure to follow the removal of a growth from the middle turbinate bone and great improvement, if not cure, when local treatment for the naso-pharyngeal mucous membrane has been adopted.

The slow pulse (infrequent pulse—bradycardia) in the subject of arterial atheroma is often, though by no means invariably, a grave sign. It is a more dangerous symptom than the irregular pulse and than the rapid pulse except in its extreme forms. A pulse at the rate of 60 and less is not very common; a pulse of 40 is rare, although such may coexist with perfect health. It may be taken as a rule that a pulse of 40 indicates a condition of some gravity; it is in some cases associated either with degeneration of the muscle of the heart or with an atheromatous lesion of the aorta. It has been thought to be pathognomonic of fatty degeneration of the heart, but this is by no means proven; in such cases the pulse may be very rapid, and running until the moment of death. Broadbent says that the infrequent pulse is far more commonly absent than present even in advanced stages of fatty change in the heart. The very slow pulse is chiefly manifested in those cases of fatty degeneration of the heart which are due to obstruction (usually from atheroma) of the coronary arteries. Baumgarten has seen it as low as 12 per minute in such a case.

Slowness of the pulse is in very close relation with some diseases of the nervous system. It is distinctly associated with epilepsy, especially with the slight forms (*petit mal*).

In observing a case manifesting bradycardia, not only should the pulse be carefully counted at the wrist but the heart's pulsations, as heard by the stethoscope, should also be counted. In some cases it will be found that the heart's contractions are just double the number of radial pulses, every alternate ventricular systole failing to produce a wave that can be felt in the artery. In other instances the rates may not correspond, the heart manifesting some irregularity. Tripier considered that the slow pulse of epilepsy was always attended by some deviation from the normal regular cardiac rhythm, and conversely that there was no deviation of the cardiac rhythm in the sense of abnormal infrequency (unless in those to whom digitalis had been administered) without epilepsy. The first of these propositions, though it expresses the general rule, is not without exception; the second is disproved by many instances which I have observed. In a senile subject who has a slow pulse, careful inquiry should be made as to whether there are any manifestations of epilepsy, however slight. It may be a question whether or no the epilepsy is the direct result of the disorder of the heart whereby the circulation within the brain is greatly modified. It appears most probable from the evidence that both the epilepsy and the disturbance of the cardiac rhythm are due to a common cause, viz., a morbid condition in certain parts of the central nervous system. In a man, aged fifty-four, with diabetes mellitus, observed during a period of two years, I have found a pulse of 24 rising to, but never surpassing, 35. In seven cases I have observed the slow pulse after influenza. In the case of a man aged forty-six, an agricultural laborer, who had never suffered from any illness exept influenza (which laid him up for a month the year before he came under my observation) the pulse-rate was as low as 19 and never rose above 33. There were attacks of faintness with epileptoid signs (*petit mal*). The patient greatly improved but died suddenly after having left the hospital. In another man, aged seventy-five, observed three months after an attack of influenza, the pulse-rate was 36. In some cases the bradycardia occurring after influenza was periodic or paroxysmal. In a man, aged fifty-six, whose habitual pulse was about 72, I found that a few weeks after an attack of influenza, coincidently with severe epigastric pain occurring every afternoon for a week and lasting for three or four hours, the pulse-rate became reduced to 48; afterward when the pain was relieved the rate rose to 92 per minute. In another case of a man aged fify-two the pulse of 72 became reduced to 52 during the periodic attacks of abdomi-

nal pain, subsequently rising to 88. In a third a tachycardic pulse of 120 became slowed to 54, and in a fourth one of 120 became abnormally slow at night. The evidence seems to me to show without doubt that the poison of influenza can induce abnormal acceleration, irregularity, or abnormal retardation of the heart's pulsations by its influence upon the nerve mechanism of the cardiac reflex. An extreme retardation of the pulse-rate also occurs sometimes after diphtheria, evidently in association with diphtheritic paralysis and most probably due to changes in the nerves or nerve-centres. Bradycardia may also occur after other forms of infectious disease, though by no means in such pronounced forms as in influenza and diphtheria; usually the symptom disappears after a few days or in a few weeks. I have observed a pulse of 40 per minute in a woman aged fifty-six after acute bronchitis. The heart had begun to fail and œdema of the legs and of the lungs was commencing.

Injuries of the cervical vertebræ, especially fractures, have been frequently found to be followed by slow pulse. Observations showing this have been recorded by Gurlt, Charcot, Jonathan Hutchinson, Rosenthal, and others. In a case recorded by Holberton, a man, aged sixty-four, injured the cervical part of the spine by an accident in the hunting field. Two years afterward the pulse was found to be only 20. During the epileptic fits which now occurred, it sometimes dropped to 8 or even 7½ per minute. He died in one of these attacks, and at the post-mortem examination it was found that the medulla oblongata and the upper region of the spinal cord had been compressed, owing to inflammatory thickenings about the first and second cervical vertebræ evidently the result of the original injury. One of the most remarkable instances of slow heart that I have ever witnessed was in the case of a man, aged forty-five, whose pulse-rate was 38 to 40 but irregular, some of the pauses being so protracted that it seemed as if the pulse would never return. These long pauses lasted five seconds and were followed by groups of three (on some occasions two) pulsations. The pause, during which the heart was absolutely still, was nearly twice the duration of the group of three systoles. After some observation of the case a diagnosis was made of aneurysm of the aorta. After death a dilatation of the descending aorta was found and both the vagi nerves were implicated in a cancerous mass. It was evident that direct irritation of the vagi was occasioned by the malignant growth.

From these data it seems probable that the abnormal retardation of the heart's pulsations is essentially due to causes primarily affecting the nervous system, that these lesions are chiefly in the medulla oblongata, about the roots of the vagus and in the upper part of the

spinal cord; but there may be such involvements of the vagus itself or the ganglia of which its fibres are components that reflex effects are produced upon the cardiac nerve-centres. The immediate expression of such disturbances, whether direct or reflex, is an irritation of the vagus or a state in which its action is relatively in the ascendant, so that its controlling power over the heart is abnormally manifested. It is an unsolved question whether in the cases where there is actual atheroma of the coronary arteries in cases of bradycardia, the cause of the symptom is a reflex transmitted from the diseased area of the vessel to the nerve centres, or whether there is an undetermined coexistent central affection. At any rate it seems most probable that the dilatation of the heart-chambers and the degeneration of the muscular elements in the cases in which no coronary atheroma exists are secondary to the nervous implications.

It must not be concluded that all cases of senile bradycardia are of dangerous import, though those which are attended with cerebral disturbances or with concurrent organic affections of the heart undoubtedly are so. I have observed in an apparently healthy old man of seventy-eight, who had some gouty tophi in the right ear and whose right radial artery presented undoubted signs of atheroma, a pulse of 36 to 40. It had previously varied between 28 and 32. There were no abnormal signs to be found on examining the heart, and two years after my first observation it was reported to me that the old man was in good health. The slow pulse may be hereditary. I have observed a pulse of 44 to 48 in a generally healthy lad of eighteen whose father was said to have been the subject of slow heart.

In regard to the treatment of cases manifesting slowness of the pulse, if there be no concurring morbid signs, cerebral or cardiac, caution should be given against physical overstrain and excess of every kind, but medicines may be entirely unnecessary. The therapy of those cases in which there are such concurrent morbid signs is not very satisfactory. The treatment for dyspepsia and for the gouty condition may be put in force. The only drug which has any effect in increasing the heart-rate in my opinion is belladonna in doses of 10 minims of the tincture or half a grain of the extract three times a day, interrupted at intervals of four to seven days and whenever dryness of the throat is complained of. Atropine is well known to antagonize the inhibiting power of the vagus on the heart in the dog and the rabbit, though the drug has far more effect in the former animal than in the latter. Von Bezold has found that in man it can markedly increase the rate of the heart's pulsations. Stimulating liniments rubbed in over the spine or counter-irritations in the form of sinapisms have, I think, some influence for good in the treatment of

bradycardia. I have seen an increase of the rate of the pulse follow systematic galvanization of the spinal cord, one pole being placed just below the occiput and the other in the lower dorsal region.

I will now assume that the patient who shows some signs of atheroma of the systemic arteries complains of *pain at the heart.* Pain within or near the præcordial region may accompany any of the disorders of the heart-rhythm that we have considered, but it may be manifested *per se.* The first practical question is: Is it of dangerous import or no? The note of danger is sounded if it has the character of angina pectoris. In a very considerable number of cases the occurrence of angina pectoris is the signal that there is atheromatous disease of the coronary arteries. It is not so in all, for there may be angina pectoris without coronary blocking, and conversely disease of the coronary arteries without angina. We shall touch upon this point later on in our review of the subject of arteriosclerosis. Our present purpose is to determine whether the painful symptoms which our patient presents should retain him in the group we are now considering, or whether they should relegate him to Group II. of cases which we believe to present disease of the coronary arteries.

The majority of cases of pain at the heart are not examples of true angina pectoris. The pain in the senile subject of atheroma whose coronaries are not diseased is usually the concomitant of dyspepsia. There is gastralgia or cardialgia. The seat of pain is especially the epigastrium. The differential diagnosis is to be made by a consideration of the associations—flatulence, weight at the epigastrium, acidity—these symptoms being interchangeable; thus a patient of Ewald's described himself as being "sometimes a vinegar factory and sometimes a gasworks." Neuralgia may affect the intercostal nerves. In such cases there are usually local foci of intensity of the pain and tender points about the sternum, the vertebræ, and the mid-axilla. In pleurodynia, which is probably an affection of the pleural nerves, there is no such localization. In herpes zoster some pain may be manifested before the appearance of the eruption as well as for considerable periods afterwards. Neuralgia in the heart region may occur from neuritis in the subjects of gout or of diabetes.

In sequence to influenza there may be severe and intense pain, simulating angina pectoris, referred to the area occupied by the heart and aorta or to the sternum. In most of the cases I have observed the constant, as distinguished from the paroxysmal, pain has been of a dull aching character, but subject to exacerbations. The parasternal pain was exceptionally as of a knife cutting; in a small minority a tender spot was found close to the sternal border. The pain extended down the left arm in three instances, in one of these the dis-

turbed sensation being described as a deadness; in another case it was severely felt in the back. In many instances the suffering was so precisely felt in relation with the heart that the patients could not be convinced that there was no organic disease. The pulsations were accompanied by pain and by most uncomfortable sensations, though the rhythm was normal and tranquil, and the heart sounds well pronounced and in their due relations one to the other. In certain of the cases manifesting paroxysmal pain the symptoms closely approached those of angina pectoris. In one, five months after influenza, there occurred attacks of intense præcordial pain as of a "grip" or "screw," which in a moment caused the patient to fall prostrate and for a brief period completely unconscious. In another case attacks of intense pain localized in the second left intercostal space—where was a tender spot—were initiated by slight exertion. In a third, paroxysms of pain at the heart, of much less severity than those in the former instances, had occurred at intervals ever since an attack of influenza, and in these seizures the patient had the feeling of impending death. Such feeling was graphically described by another patient "as if his heart were a pendulum suddenly stopped at one swing." In several cases there was loss of consciousness. Such severe symptoms can occur after influenza at any age, but they are of more serious import in the senile subjects of arterial atheroma.

In regard to the pathology of cases of pain at the heart which are not of the nature of angina pectoris, we can roughly group them in the class of neuralgias. In cases of dyspepsia, especially gouty dyspepsia, we may consider them as reflexes from irritation of the gastric mucous membrane. I have observed distinct deposits of urates among the gastric tubules in a case of gastric catarrh. It would seem to me most probable that in many other cases the morbid condition of the finer nerve branches may be in the pericardium, the pleura, or the ganglia in the neighborhood of the heart itself. After influenza there is sufficient evidence to show the probability that there may be a distinct neuritis in these situations, and neuritis may occur with gout or diabetes. In cases of acute aortitis, which we have already considered, it is in the highest degree probable that the symptoms of suffering are due to the involvement of the disease of the nerves and ganglia which are in such close contact with the wall of the great artery.

In regard to the therapeutics of such cases, the minor forms may soon yield to the treatment advocated for dyspepsia in the subjects of gout, or to mild local counter-irritation by sinapisms or friction with liniments or ointments containing opium, belladonna, aconite, or menthol. As agents administered internally for the relief of

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pain, I think that the milder analgesics, phenacetin or antipyrin, should be tried before opium is had recourse to.

In the more severe forms, with rest and local counter-irritation, the hypodermic injection of morphine hydrochlorate, gr. $\frac{1}{4}$ to $\frac{1}{3}$, is often necessary for relief. Diffusible stimulants (ether, ammonia, valerian, sumbul, or musk) should be administered at the time of each injection. In many cases I have found quinine in 5-grain doses, dissolved with 20 minims of dilute hydrobromic acid in an ounce of water, administered every four hours, relieve the pain and improve the condition, the action being better than that of opiates. The continuous galvanic current from the spine to the seat of pain has also been very useful.

We will now consider—the former questions being eliminated—whether the patient is to be placed in Group III., in which are cases of *atheroma of the coronary arteries*. The criteria on which we depend for assigning the case to this group are, (*a*) the painful signs of angina pectoris with the manifestation of general thickening of the arteries and abnormal tension of blood within them; (*b*) the occurrence of dyspnœa on exertion and the other symptoms which we have already considered as indicating obstruction to the due arterial blood supply to the heart. The chief points on which the observer should rely as indicating an attack of *true angina pectoris* are the following: 1. The principal seat of pain is the middle or lower part of the sternum, somewhat to the left; it may extend to the axilla and the back, or may radiate up to the neck, the lower jaw, or the occiput. In many cases the pain extends down the left arm to the elbow or the fingers; in these situations it may be accompanied by a sensation of coldness or numbness. It may pass downward to the lower part of the abdomen or to the testicles. It is not increased by external pressure. Exceptionally it may radiate to the right side, involving the right arm. 2. The pain referred to the sternum is accompanied by a sense of constriction. The suffering is always severe, and in some cases an indescribable torture, perhaps the most severe form of pain endured by human nature. 3. The patient, when the attack occurs, becomes motionless in one position. There is no marked dyspnœa, but breathing may be shallow, or even arrested. 4. The face is usually pale, or of leaden hue and bedewed with a cold sweat. The patient waits with intense anxiety the end of the nerve-storm. 5. There is an extreme sense of depression and feeling of impending death; but the attack once over, the patient experiences a sense of free relief, and there are not frequent repetitions of pain, as in dyspeptic and hysterical cases. In some persons the attack is followed by flatulent eructations or vomiting.

Instances have been recorded in which the attack has commenced with pallor, attended by coldness and stiffness in the limbs—the oppression and pain at the heart, of comparatively slight intensity, following—the vaso-motor angina of Nothnagel.

The sign which differentiates true from false angina pectoris in the most important degree and most practical manner is the general firmness and thickness of the arterial walls (to be distinguished from the local patches of atheroma), and the state of abnormal tension of the blood stream within the arteries. Instances of fatal angina have occurred when these signs were wanting, but they are extremely rare. In dangerous and fatal angina pectoris there is in the majority of cases a coexistence of disease of the coronary arteries with such abnormal contraction of the muscular walls of the systemic arteries as to reduce almost to zero at the time of the paroxysm the column of blood transmitted by them and thus lead to ischæmia of the heart and of the great nerve centres.

The treatment of angina pectoris by arterial relaxants will be described in the section on arteriosclerosis.

Obstruction of the coronary arteries may occur without the manifestations of angina pectoris. The first note of the obstruction to the due arterial supply to the heart is breathlessness on exertion. With this symptom there may be oppression and sense of constriction of the chest, with more and more acute pain as there are approaches to the true angina pectoris. The gradual dilatation of the left or the right chambers may be traced as the left or the right coronary artery is blocked. There are at first occasional faintnesses and afterwards increasing feebleness. Gradually dropsy of the lungs or of the lower extremities, perhaps general anasarca, follows. In some cases the signs of dilatation of the aorta or of disease of the aortic valves (obstructive or regurgitant or both combined) give the clew—the atheromatous affection of the great vessel and its valves encroaches over the mouth of the coronary artery. In the absence of the physical signs of these affections we may infer, when the symptoms I have just enumerated develop, that there is, in the subject of atheroma of the systemic arteries with whom we are now dealing, chronic arteritis of one of the coronary arteries or of both these vessels. The treatment of cases manifesting such symptoms I have already indicated in considering the therapeutics of chronic aortitis.

Syphilitic Disease of the Arteries of the Brain.

The thickenings of the walls of the cerebral arteries which are met with in the subjects of syphilis differ in many points from the athe-

romatous patches observed in the old and in the gouty. The thickenings take the form of nodular swellings much more localized than in ordinary atheroma; they are firm and fibrous but very rarely calcareous. The late Dr. H. G. Sutton said: "When I have at a post-mortem examination found syphilitic changes in the cerebral arteries the appearances have been such as we could not mistake. There has been a very limited patch of thickening in the adventitious coat of the vessel, making a grayish tumor projecting from the vessel which has immediately rivetted our attention. Its limitation at once led us to suspect that it was syphilitic, the other vessels being normal." On cutting into the mass it is found that the thickening extends through the wall of the artery, and there is some yellow gummous material among the fibrous tissue. The intima is roughened by the growth and blood coagulates upon it and plugs the vessel; the portion of brain supplied by the artery becomes softened. Dr. Sutton adds: "I have known such a plug to spread across to the opposite cerebral artery and lead to a double softening. The cases I have seen have been plugging of the middle cerebral artery, but the disease may affect the basilar and other arteries." [14] These views of a shrewd and long practised observer confirm those of Lancereaux and Baumgarten that the syphilitic lesions of the cerebral arteries only superficially resemble atheroma. The vessels most frequently affected are the internal carotid, middle cerebral, vertebral, basilar, and posterior cerebral arteries. Usually more than one artery presents the changes. Syphilis chiefly attacks the larger arteries of the brain and those of the circle of Willis. The disease sometimes commences as a periarteritis; it is then first manifested in the lymphatic tissue, which surrounds the vessels, as minute isolated yellowish nodules. The external coat of the artery becomes infiltrated with cellular bodies, the middle coat is invaded by the swelling and becomes atrophied, and the tube of the artery becomes obliterated by the increase of the thickened material, or a coagulum forms as just now described. The nodular thickenings are due to a fibro-nuclear growth, the structure of which resembles a syphiloma as found in other places. In some cases, however, and in some situations the arterial coats may be dilated—so there may be in the same subject a thickening of the arterial coats at one spot and a thinning of them at another, the vessel being contracted at some parts and dilated at others.

Charrier and Klippel (*Revue de Médecine*, September 10th, 1894, p. 775) describe the syphilitic affections of the cerebral arteries as of three forms: (1) Periarteritis (artérite de voisinage), which occurs as gummatous nodules in the tissues external to the artery and may even leave the latter intact, though more often it induces consecu-

tive disease of the coats of the vessel; (2) gummatous arteritis, in which gummatous deposits have their seat in the walls of the vessel, the lesion commencing sometimes in the external and at others in the internal coat; (3) sclerous arteritis, which occurs in the basilar artery and in the carotids, is generally circumscribed, and appears as slightly elevated patches or as round and hard spots along the vessels, causing bulgings both of the external and internal coats. The transformation in this case is fibrous but not calcareous. In some instances this form of lesion commences in the internal coat, as formerly described by Heubner.

The lesions consecutive to syphilitic endarteritis may be (1) an obstruction of the vessel, as from obliterative endarteritis, (2) thrombosis within the vessel, (3) rupture inducing intracranial hemorrhage, or (4) aneurysm.

It is well established that aneurysms of the larger arteries of the brain are frequently due to syphilitic disease. The smaller arteries within the cerebral substance are occasionally affected by syphilitic changes.

Syphilitic endarteritis affects males more frequently than females It is chiefly a consequence of the acquired disease. According to Gowers about fifty per cent. of the sufferers are between thirty and forty years of age at the time of the manifestation and less than twenty per cent. between forty and fifty; over the age of fifty the disease is very rare. One-third of the total number are between twenty and thirty. Of 26 cases 16 occurred within six years of the infection, 6 during the second six years, and 5 during the third six years. The affection may, however, be a manifestation of syphilis of the infant, for Chiari (*Wiener medizinische Wochenschrift*, 1881) has described a case, in a child of fifteen months, who presented syphilitic manifestations six weeks after birth; the post-mortem examination showed syphilitic endarteritis of the basilar artery with thrombosis of its whole trunk. Gowers also has noted the case of a boy, aged 8, the subject of inherited syphilis, who died from cerebral hemorrhage, and at the autopsy there was found syphilitic disease in the vertebral and cerebellar arteries (Gowers, "Manual of Diseases of the Nervous System," Vol. II., G. and A. Churchill, 1888, p. 356).

It has been thought that syphilitic disease of the arteries arises in the majority of cases at a remote period from the original infection, but this is no doubt an error. Thibierge has said (*Gazette des Hôpitaux*, January 26th, 1889) that from its very earliest phases syphilis manifests its morbid action upon the arteries. Hughlings Jackson, Lancereaux, Wilks, and many other observers have insisted that the majority of the manifestations of cerebral syphilis have been

due in the first place to a syphilitic disease of the arteries. Grault recorded a case in which eight or nine months after infection the post-mortem examination demonstrated cerebral arteritis which had caused meningeal hemorrhage, and a patient of Millard died of cerebral arteritis five and a half months after the initial syphilitic lesion. Gaudichier reported 38 observations of cerebral disease arising twelve or fifteen months after infection. In a recent discussion at the Royal Medical and Chirurgical Society of London many similar experiences were mentioned. Althaus narrated a case in which eight months after infection there were signs of syphilitic disease of the cerebral arteries that yielded to appropriate treatment, and another in which headache, convulsions, and hemiplegia—signs in all probability indicating syphilitic cerebral arteritis—developed three months after infection. In a somewhat similar case recorded by Kahler extensive disease of the arteries of the base of the brain was seen at the post-mortem examination. Jonathan Hutchinson has said that many, perhaps most, of the affections of the nervous system in the secondary period of syphilis are due to disease of the blood-vessels, and under this head we may include extensive implications of the minute arteries of the pia mater, whether of the brain or cord.*

Dr. G. Ogilvie (*Lancet*, June 1st, 1895) gives a valuable summary of Naunyn's statistics, showing beyond doubt that syphilitic diseases of the nervous system appear more frequently during the first year after infection than in subsequent years, though about three or four per cent. are not manifested till after the twentieth year. They are even more frequent during the first six months of the first year than in the subsequent half year.

The tables show that the prognosis is considerably more favorable in cases where treatment has been begun early—before the end of the first four weeks.

In some cases the history of antecedent syphilis is very obscure; usually for diagnosis we have to rely chiefly on the symptoms, but the scars of old cutaneous syphilis should be sought for and the previous history closely scanned.

Symptoms and Course.—In syphilitic disease of the arteries there is usually a series of symptoms preceding the graver manifestations. Prominent among these is headache (or head pain). If a patient who has never been the subject of headache in his former life begins to manifest such symptoms, the possibility of intracranial syphilis must not be overlooked; and if the suffering be persistent for days

* "Affections of the Nervous System in the Early (Secondary) Stages of Syphilis," Royal Medical and Chirurgical Society of London, February 26th and March 12th, 1895.

or weeks the probabilities are greatly increased. It has been said that, except in some forms of intracranial tumor, there is no form of headache so lasting as that of cerebral syphilis. In syphilitic disease of the arteries the headache is frontal or fronto-parietal, diffuse, and constrictive, rendering the sufferer restless and hypochondriacal; while in gummatous syphilitic tumor it is deep-seated and circumscribed, as if caused by a nail driven in. The absence of optic neuritis (for this never occurs in the arterial disease alone) serves to differentiate from tumor.

Such headache in syphilitic arteritis may be the only notable symptom for a long period. It is often worse in the evening or at night. Giddiness may be associated with the symptom. Then may follow signs of weakness in certain muscles, or stiffnesses and spasms, numbnesses or tinglings, difficulties of speech, misplacements of words—aphasia more or less pronounced—some mental obscurations, incoherence, or a tendency to somnolence. Some of these signs, apparently slight, are nevertheless very important for diagnosis. For example in the ordinary speech on one occasion a single word may be forgotten or misapplied, a short time afterward the *same word* again becomes a difficulty; or sensations of "pins and needles" or signs of muscular enfeeblement may recur many times in the same part of one of the hands or arms. The first occurrences are often thought to be insignificant, and may be of no moment, but the *re*currences are of much diagnostic importance. It is a more crucial sign if there be a local paralysis or monoplegia.

In some cases the slightly pronounced premonitory symptoms are, after a time, followed by a sudden and fatal apoplexy, and this is in some instances due to the rupture of a vessel in the membranes of the brain, in others to the sudden blocking (thrombosis) of the basilar artery or of one of the branches coming from the circle of Willis. In many cases the stroke of apoplexy caused by the plugging of the diseased vessel is not followed by sudden death but by a lasting hemiplegia.

In syphilitic disease coma is the exception; consciousness, as Gowers says, is more frequently preserved than lost. Successive manifestations of paresis afford strong presumptive evidence of syphilitic arteritis. For instance, a patient may complain of weakness of one leg; in a day or two the arm of the same side may be enfeebled, a few days afterward there may be complete hemiplegia with the accompanying facial paralysis (of the same side) and aphasia —if the lesion be in the left hemisphere, the paralysis being on the right. In some cases the lesions disappear, useful power being regained, because there is an incomplete blocking of the artery, and

after the early effects of diminished supply of arterial blood and of temporary congestion the circulation in the disturbed area may be re-established. The restoration in one part, however, may soon be followed by new lesions in another; there may be paralyses in the limbs hitherto unaffected.

In some cases *aphasia* is the sole declaratory sign of syphilitic arteritis. It may be very transitory, probably noticed more by others than by the patient himself—a recurring but unwonted difficulty in finding a given word or a repeated misapplication of a word or two. Afterwards there may be increasing difficulties up to a total inability to express ideas in language or in writing.

Another not infrequent mode of manifestation of syphilitic intracranial arteritis is *facial paralysis with arm paralysis of one side followed by a similar paralysis of the opposite side*—the lesion is symmetrical. The diagnosis from such symptoms of syphilitic arteritis as a cause is almost certain.

MM. Charrier and Klippel classify the chief forms of manifestation of syphilis of the cerebral arteries as four: (1) the form of *fatal apoplexy*, the disease causing ruptured arteries of the meninges or of the base of the brain; (2) the form of *grave paralysis*, causing lasting hemiplegia—generally due to obliterating arteritis with consecutive necrosis of the affected area, but in some cases to hemorrhage from ruptured miliary aneurysms; (3) the form of *slight aphasia* and *transitory, varying paralyses*—the group most characteristic of the ordinary syphilitic arteritis; (4) the form of *intellectual involvement*, presenting many of the signs of general paralysis of the insane but differentiated from the latter by the premonitory symptoms and by the coexistence with other of the forms of paresis, as in Group III.

In doubtful cases a vigorous antisyphilitic treatment is justifiable even as a means of diagnosis. In all the forms, except the sudden and complete apoplectic form and the intellectual form, there may be great improvement, if not cure.

Treatment.—If from the symptoms already indicated the diagnosis is correctly made of syphilitic disease of the arteries, the physician has the satisfaction of knowing that that treatment is very frequently effectual. There are, however, limits to the possibility of therapeutic means becoming successful. In the rare cases in which sudden apoplexy occurs in a syphilitic subject there is most probably the bursting of an aneurysm of a cerebral artery; not invariably, however, for there may be thrombosis of the basilar and the arteries proceeding from it; in these cases treatment is for the most part hopeless. If the syphilitic disease has caused thrombosis of the basilar

or internal carotid, speedy death is almost certain and obstruction of the trunks of both middle cerebrals is almost always fatal.

Next in gravity is obstruction of the vertebral, and next that of the middle cerebral of one side. In general the danger to life is far less in plugging of the artery by clot or by syphilitic obstructive disease than in senile softening.

The most important drugs in the treatment of syphilitic disease of the arteries are the iodides—especially the iodide of potassium. The treatment should be prompt, for it can cause the removal of the disease from the wall of the artery and prevent repetitions of the morbid process in other cerebral arteries. It should be put in force if syphilitic disease is only suspected, for if the signs of rapid recovery follow it may be a means of diagnosis. Gowers recommends that the iodide should be given in doses of 10 or 15 grains every six or eight hours or administered in scruple doses by the rectum. In the event of no signs of improvement being manifested in the course of a week, I would advise that the drug be omitted for a few days. The administration of mercury by the mouth or by inunction is considered by some to be of much less importance. Gowers says, "It may be well also to rub in some mercury, especially if the symptoms indicate extensive disease." Charrier and Klippel recommend an energetic treatment. They advise that on the first day of treatment the patient should be given 6 grams (92½ grains) of the iodide, on the second day 8 grams (123½ grains), afterward 10, 12, and up to 16 or 18 grams (154 increased to 277½ grains) in the twenty-four hours. Thus the dose each day progresses from a minimum of 1½ drachms to a maximum of rather more than 4½ drachms. Concurrently inunctions of strong mercurial ointment are to be practised. At first the ointment should be rubbed into the upper arm above the elbow, then the front of the thighs, next the calves, and afterward the groins; each rubbing should be for ten minutes at least, and the earlier frictions, at any rate, should be done under the eye of the physician. It is recommended that the part rubbed should be covered by an ointment-smeared flannel to be left on the surface till the next day's inunction. Three times a week the patient should have a sulphur bath, the maximum temperature of which should be 34° C. (93° F.) and the duration twelve to fifteen minutes. It should be insisted that the patient brush the teeth and clean the mouth carefully eight or ten times a day with a gargle of chlorate of potassium. With these precautions it is said that the treatment can well be tolerated and that the results are extremely good in cases wherein a less energetic treatment has been tried in vain (*Revue de Médecine*, September 10th, 1894, *La Semaine Médicale*, October 3d, 1894).

It may be judicious thus to "frapper fort et frapper vite," but the judicious physician will interrupt the treatment if signs of general depression or of mercurial poisoning become evident. The importance of prompt and sufficiently energetic treatment cannot be overestimated, but it is well that we should realize the possibilities. So far as the symptoms in the given case depend upon obstruction in an artery from syphilitic disease encroaching upon its lumen, recovery can be perfect. Moreover, the means adopted can prevent the occurrence of disease in other arteries, for we know that many arteries may be involved or one artery may be affected in many spots. Nevertheless we cannot, by treatment, however energetic, restore the status quo ut antea if there has been in consequence of the disease thrombosis of the vessel and softening of the brain. It is important to bear in mind what Gowers has said on this subject. "By antisyphilitic treatment we may remove the disease in the wall of the occluded vessel, we may prevent an increase in the symptoms, but we cannot remove the clot that has finally closed it and has extended on into the distant branches. We cannot alter by our treatment the process of softening." "The syphilitic origin of the disease does not influence the prognosis of developed palsy. Most cases improve and many recover, but they improve and recover in the same way as in every other form of acute cerebral lesion—because the symptoms are of indirect and not of direct origin and sometimes in obstruction, because a collateral circulation is possible. If the softening involves the motor path or centres, enduring hemiplegia is the result. . . . Because a palsy is due to syphilis it is often assumed, as a matter of course, that it will be removed by anti-syphilitic treatment. The assumption is correct as regards the pressure-effects of syphilitic growths and many syphilitic affections, but it is not true of necrotic softening from vascular disease. I have seen many patients who had been assured because their hemiplegia was of syphilitic origin that they would certainly be cured, and when after a year or two the paralysis remained, they were naturally indignant at the erroneous opinion they had received" (Gowers, "Diseases of the Nervous System," Vol. II., 1888, p. 409).

Arteriosclerosis—Arterio-Capillary Fibrosis.

The affection we are about to consider is more general, or much more widely spread throughout the arterial system, than atheroma. Arteriosclerosis may exist without atheroma and atheroma without arteriosclerosis, though frequently both are manifested in the same subject. Abnormal thickening may be demonstrated in the larger as well as the smaller arteries, especially in chronic Bright's disease,

but the smaller arteries are in the greatest degree affected. Recent researches made by Dickinson show that in cases manifesting granular kidneys the aorta and the innominate and the femoral arteries are all increased in the total thickness of their walls, in the thickness of their muscular coat, and in their circumference. The smaller systemic arterioles have some special cause of hypertrophy beyond that which affects the larger. In chronic renal disease, Dickinson says, "There occurs a hypertrophy of the cardio-arterial system which is universal from its origin to its termination, and comprises not only the ventricles and the arterioles, but affects also the intermediate arteries of every size" (*Lancet*, July 20th, 1895). The morbid changes have been demonstrated in the minute arteries and larger capillaries of the pia mater, in the retina, in the arterioles of the kidneys, the heart, the lungs, the spleen, the stomach, the brain and spinal cord, and the skin, as well as in other situations.

Morbid Anatomy.

Arteries of the Pia Mater and Systemic Arteries.—The morbid changes can be most readily demonstrated in the arterioles of the pia mater. In these microscopical evidence leaves no room for doubt that the arterial wall can be so thickened that its linear measurement is more than twice that of a corresponding artery from a healthy subject. As to the nature of this thickening and the relative involvement of the normal structures of the artery, there has been much difference of opinion. Sir George Johnson, about the year 1867, was the first to describe a thickening of the minute arteries from various tissues in cases of chronic Bright's disease which he looked upon as a genuine hypertrophy of the muscular coat of these vessels and considered to be in constant relation with hypertrophy of the left ventricle of the heart. On these observations he founded a theory which will be subsequently alluded to. The late Sir William Gull and Dr. Sutton in 1871 published a series of researches showing that in chronic Bright's disease with granular contracted kidney the small arteries (those of the pia mater being for the most part subjected to examination) as well as the capillaries had undergone a change which they denoted as a hyaline-fibroid transformation. The degree in which the affected vessels were altered and the extent of the morbid change throughout the arterio-capillary system varied greatly in the cases subjected to examination. The diseased vessels were frequently tortuous and their lumen was lessened, the internal coat was sometimes markedly thickened and presented in its external layers a finely fibrous or a molecular arrangement. The muscular coat was often relatively increased, but in some parts it was wasted and degen-

erated; hypertrophy of the muscular elements was by no means constant. External to this was often seen a more or less homogeneous, hyaline formation and with it, in some portions, a coarse fibroid and granular material. The change was seen to occur chiefly outside the muscular coat. The capillaries presented a hyaline-granular change.[15]

Dickinson also found that in chronic Bright's disease the affected vessels, usually the smaller arterioles, were often thickened in a sufficiently striking manner, the thickening involving both the muscular and fibrous coats. Degeneration of the muscular coat was distinctly seen; the arterial sheath also had become wavy and presented an appearance as if swollen with translucent structureless exudation. Lionel Beale, than whom there cannot be a more competent microscopist nor a more careful observer, found that the condition of the thickened arterial coats was not one of true muscular hypertrophy—the contractile tissue had become degenerated into mere fibrous tissue. It was the connective tissue that was often enormously thickened, a new material being deposited externally to the muscular coat as well as, probably, internally to and among the muscular fibre cells; thus the lumen of the artery was encroached upon and the current of blood through it of course obstructed.

Arteries of the Kidney.—In the minute arteries of the kidney Gull and Sutton found in the early stages of chronic Bright's disease an excess of fibrous tissue. This fibrous change was chiefly in the external coat where there was fibroid hyperplasia; the muscular coat did not appear to be increased. The visible morbid changes were considered to be due to the primary formation of fibroid or hyaline-fibroid substance in the intertubular parts of the kidney including the vessels, the tubular and intertubular structures of the organ becoming consecutively atrophied. The formation commenced in different parts of the kidney, commonly near the surface, but also in the outer coats of the arterioles and in the capillaries. The changes in the minute renal arteries were precisely similar to those observed in the arterioles of the pia mater and of other parts of the body. There can be no doubt from subsequent observations that the arterioles of the kidney in the granular contracted form of renal disease present thickenings both of their external and internal coats. The fibrous proliferation in the external coat is continuous with that which occurs so abundantly in the connective tissue of the kidney itself. In 40 post-mortem examinations in cases of chronic renal disease, Loomis found, as regards the systemic and renal arteries, the external coat thickened in many cases; in 21 cases this fibrosis was not to be distinguished from that of the neighboring interstitial tissue and about the glomeruli; the internal coat alone was diseased

in 12 cases, and the muscular coat was thickened in 5 cases. The morbid changes are external to the arteries as well as in their walls.

Arteries of the Heart.—Gull and Sutton found the minute arteries in the walls of the heart much thickened by the formation of hyaline-fibroid substance. A special study of arteriosclerosis of the heart has been more recently made by Huchard. He finds that the arterioles within the myocardium in the ventricular wall or in the musculi papillares may present the signs of obliterating endarteritis—their internal coat becomes thickened and the thickening may increase by stages to the complete obstruction of the vessel. The muscular fibres supplied by the affected vessel undergo necrosis; granulations beset them and bring about their fragmentation; then the fibrous interstitial tissue begins to proliferate. Finally the affected patch of muscle is replaced by a tissue which becomes more and more fibrous. There may be, however, a periarteritis as well as an endarteritis. The external coat of the arterioles may be thickened and may send out prolongations of fibrous tissue among the muscular elements.

Many authors—among them Bristowe, Friedreich, and Lancereaux—believe that the chief changes are outside the vessels in the general connective tissue of the heart. There can be no doubt, however, that fibroid changes in both the internal and the external coats of the arterioles can be demonstrated in cases of hypertrophy and dilatation of the heart, though an independent proliferation of the connective tissue may be associated therewith. The morbid changes in the heart are strikingly analogous to those which occur in the kidney in chronic Bright's disease.

Arteries of the Lungs.—Endarteritis and periarteritis have been abundantly demonstrated in the pulmonary and the bronchial vessels. According to Boy Tessier the arteriole may be obstructed by a series of swellings in its internal coat, these being isolated from each other; or it may be narrowed by a ring of thickening in the manner of obliterating endarteritis. These lesions are commonly to be demonstrated in emphysema of the lungs occurring in old persons and in the subjects of chronic renal disease. It is well known that vesicular emphysema of the lungs and granular contracted kidneys often coexist. Gull and Sutton found the kidney lesion in 22 out of 33 cases of emphysema in persons about middle age.

Arteries of the Retina and of the Central Nervous System.—Bader observed in the small arteries of the retina and the choroid a thickening of the walls by a homogeneous, strongly reflecting, not quite transparent substance; this was in fact a corroboration in regard to these arteries of the views of Gull and Sutton. In the retinal arteries Brailey and Edwards found that there was a constant thickening in

chronic Bright's disease even when no abnormal appearances had been observed with the ophthalmoscope during life. The thickening was chiefly of the inner coat and it could progress to obliteration of the vessel (Transactions of the Ophthalmological Society of London, Vol. I., p. 44). Both the arteries and the capillaries may present aneurysmal dilatations. The hemorrhages so frequently observed in cases of granular kidney (albuminuric retinitis) are due to local softenings of the arterial wall or to the bursting of minute aneurysms. Consequent upon these alterations in the arteries and upon the hemorrhages are degenerative changes—fatty degeneration of the nerve fibres, infiltration with round cells, and separation of the fibres by hyaline fibroid material.

In the *spinal cord* Gull and Sutton showed (in 1877) that the arteries and capillaries might present thickenings and fibroid changes of like kind with those seen in the vessels of the kidney, but there was found also a homogeneous and granular material diffused through the cord structures in defined patches. A fibroid change was also observed in the cord in no obvious relation with the vessels, being most marked where the ordinary connective tissue was most abundant. The changes in the arteries and in the connective tissue in the cord closely resembled those observed in the like tissues in the kidney of interstitial nephritis; but fibrosis of the cord may occur coincidently with fibrosis of the kidney, or be in advance of the fibroid change in the kidney, or occur as part of a general fibrosis altogether independently of renal disease.[15] It has been considered probable that the determining cause of the sclerosis of the posterior columns of the cord, in a large proportion of the cases of tabes dorsalis, is a syphilitic endarteritis.

A distinct endarteritis in the meningeal arteries proceeding to the posterior portion of the spinal cord has been demonstrated by Hippolyte Martin. A similar change affecting the brain and membranes may be a cause of general paralysis of the insane.

Pathological Associations.

The most important is that with *hypertrophy* and *dilatation of the heart.* It was noted by Bright himself that chronic renal disease is often accompanied by hypertrophy of the left ventricle of the heart, and the observation has been confirmed by all subsequent observers. Cardiac hypertrophy or enlargement has been found to occur in from about fifty to eighty per cent. of all cases manifesting contracted granular kidney.* Clinical observation convinces us that the hyper-

* Forty-six per cent., Grainger Stewart; forty-eight per cent., St. George's Hospital records on excluding certain complicated cases; seventy-four per cent., Dickinson; sixty per cent., Loomis; eighty per cent., Guy's Hospital (Galabin).

trophy is manifested quite early in the disease. The forcible heaving of the left ventricle may be felt and the tension of the blood within the arteries may be found to be abnormally great before there is any direct evidence of disease of the kidneys. Bright enunciated the theory that this hypertrophy of the left ventricle was occasioned by the fact that the blood, impure because of defective elimination by the kidneys, circulated with difficulty through the general capillaries—the heart's muscle increased in bulk and in force to overcome this obstruction. Sir George Johnson in 1867 held that the hypertrophy of the muscle of the heart was directly associated with hypertrophy of the muscular coat of the arteries; the augmented contractile power of the smaller arteries being manifested in order that these might (the increased muscular contraction narrowing their lumen) keep back the impure blood from the tissues. The heart became hypertrophied in its effort to overcome the obstruction constituted by the contraction of the muscle of the arterioles. The persistent over-action of the muscular tissues, both cardiac and arterial, was registered after death in a conspicuous and unmistakable hypertrophy. This was the "stopcock theory" which now finds few adherents, though it is adopted by Sir William Broadbent with the very significant modification that the obstruction is *not* primarily constituted by the muscular contraction of the arterioles—that is to say, the stopcock action of these fails to explain the condition—"nothing could be more clear than the demonstration of the increase of muscular fibre-cells in the thickened arterioles of chronic Bright's disease; and *except that in my opinion the obstruction is primarily in the capillaries and the arteriole contraction secondary to this*, Dr. Johnson's theory commands my entire adhesion."[16] The researches of Gull and Sutton (in 1872) showed that hypertrophy of the muscular coat was not an essential part of the changes observed in the arterioles; it could be demonstrated in some situations while degenerative changes in the muscle were observed in others, but the arterioles as well as the capillaries were thickened by a hyaline and a fibroid material. These authors considered that the morbid change in the walls of the small arteries impaired the elasticity of these vessels; on account of such impairment of elasticity, a greater force than the normal was required to propel the blood through them, and the left ventricle became hypertrophied because it had of necessity to contract with greater force in order to carry on the circulation. They observed that morbid changes in the kidney were not necessarily preliminary to the thickening of the arterioles; the heart was in some cases hypertrophied while the kidneys remained healthy. They concluded that the lesions of the kidney do not constitute an

essential and indispensable part of the process of arterio-capillary fibrosis; that the morbid changes commonly but not always commence in the kidney; that the kidneys may be little if at all affected while the disease processes are far advanced in other organs, and they may undergo extreme degenerative changes without association with the cardio-vascular lesions characteristic of chronic Bright's disease.

Dickinson's observations from post-mortem evidence went to show that arteriosclerosis was a more constant change than cardiac hypertrophy in relation with chronic renal disease. The heart might show no appreciable hypertrophy and yet the arteries of the pia mater be greatly thickened; conversely the heart might be hypertrophied and those arteries remain absolutely natural; one part of the circulatory system might be affected by the hypertrophic process and not the other. There was, however, a very constant relation with fibrosis of the kidney. Granular kidney in the rare cases in which it was observed in childhood was always associated with arteriosclerosis; and even simple inflammation (nephritis) essentially affecting the tubes and renal epithelium, though also productive of some interstitial proliferation, springing from a determinate cause and running a comparatively rapid course, was constantly succeeded by some degree of cardio-vascular change, often more marked in the heart than in the arteries, but not admitting of question in either situation. Arteriosclerosis and cardiac hypertrophy were also observed to follow accidental damage of the kidney by disease, such as calculous pyelitis. Dickinson thought the conclusion was inevitable, that the renal and vascular changes were in the relation of cause and effect. His view was that the hypertrophy of the left ventricle of the heart arose simultaneously with the vascular change or even rather before it, both resulting from a common cause. This cause was, as had been foreshadowed by Bright, obstruction in the capillary circulation.

Other observers, as Cohnheim and Hilton Fagge, considered that the hypertrophy of the heart could not be explained by merely mechanical difficulties of the circulation in the general system; that enhanced force of cardiac contraction was called for in cases in which there was obstruction in the vessels of the kidney in order that a sufficient circulation might be maintained in those which remained normal and thus a compensatory excretion of solids by the urine be effected. The agency by which this change was brought about must be a modification of the nervous system, for it is a vaso-motor reflex which regulates the activity of the circulation through the renal arteries.

The theories before mentioned attempt to explain the hyper-

trophy of the left ventricle and the enhanced force of the ventricular contractions which are observed in arteriosclerosis and chronic renal disease. Other conditions, however, than hypertrophy are not infrequently met with. In a valuable communication to the Association of American Physicians in 1888, the late Dr. Alfred L. Loomis stated that his clinical and pathological studies during five years convinced him that simple cardiac hypertrophy was not to be found in a proportion of cases of chronic Bright's disease, but that the most constant cardiac changes were dilatation of the heart cavities and degeneration of the heart-walls. Obliterating arteritis, the result of a strong hereditary tendency to general arterial sclerosis, might gradually and progressively develop for years and as a consequence there would be a compensating cardiac hypertrophy; when the extension of disease involved the parenchyma of the kidneys the nutritive power of the blood could become so diminished that all the organs and tissues of the body were enfeebled and cardiac dilatation and weak heart resulted. In 7 out of 40 cases the heart was not hypertrophied, or the walls were but slightly thickened, notwithstanding the fact that there were extensive obliterative vascular changes in the kidneys. Three cases only manifested hypertrophy of the cardiac muscle without degenerative changes and in these the disease of the kidneys was confined to the vessels, while in 20 cases the heart muscle showed degenerative changes accompanying the hypertrophy and in these there was involvement of the stroma and secreting structure of the kidneys as well as disease of the vessels.

Loomis concluded that "even in the cirrhotic kidneys the primary cardiac hypertrophy has no necessary connection as cause and effect with the kidney changes, but both are a part of the general fibrosis which has its origin in a fibroid diathesis, either hereditary or acquired, the hereditary influences being far stronger than the acquired." Nevertheless "as a rule, the more extensive the obliterating changes in the renal arterioles, the greater the degree of cardiac hypertrophy."

I cannot doubt that in many cases which have been described as hypertrophy of the heart in association with chronic renal disease and arteriosclerosis there has been a false hypertrophy such as was described many years ago by Sir Richard Quain. In some cases there is a true hypertrophy of the left ventricle with thickening and hyperplasia of the muscular fibrillæ alone; in a considerable number the muscular walls of both ventricles, in unequal degrees in each, are thick and leathery, not from increase of muscle but from fibroid development and infiltration. It has not been sufficiently realized that this fibrous hyperplasia is intimately associated with local arteritis in the

ventricular walls. So long as the thickened arteries permit an adequate blood stream the hypertrophy of the muscular fibre continues; but the left ventricle becomes enfeebled and dilated when the arteriosclerosis has so advanced as to prove a real obstruction to the blood-supply. The right ventricle in some cases follows the failure of the left; in others it becomes independently dilated and sclerosed in sequence to degenerative changes in the vessels and tissues of the lungs.

Another very frequent and important association of arteriosclerosis is with *atheroma*. Dickinson found in an analysis of 250 cases of granular degeneration of the kidney atheroma in sufficient degree to call for notice in fifty-two per cent. When the granular kidney was observed in youthful subjects, careful examination of the arch of the aorta and the heart valves showed some specks of atheromatous disease —they were found in a child, aged six, in association with profuse intertubal fibrosis and in another, aged fourteen, with granular kidney. Gull and Sutton stated that they found with granular contracted kidney the aorta and aortic valves much diseased and the seat of atheromatous and sometimes calcareous changes attended with dilatation of the aorta, with or without aortic obstruction and regurgitation. In cases of atheroma of the coronary arteries of the heart, arteriosclerosis is a frequent though not a constant concomitant. The question of this concomitance has an important bearing upon the diagnosis and prognosis of angina pectoris. I have long held the opinion that it is the coexistence of arteriosclerosis and coronary atheroma that in the great majority of instances constitutes the criterion of true angina pectoris and determines the fatal result.[17] In rare cases death from angina pectoris has taken place without this conjunction. In such it seems to me most probable that there has been an acute ischæmia of the heart consequent upon a sudden spasmodic constriction of the arteries, some cause of cardiac weakness concurring. It is noteworthy that in the case of Dr. Arnold who died at the age of forty-seven in a first attack of angina pectoris, there was but one coronary artery and, considering the size of the heart, this seemed to be of small dimensions (Latham, "Diseases of the Heart," London, 1846, Vol. II., p. 377). In the great majority of cases of true angina pectoris there is abiding evidence of thickened contracted arteries; crises of pain are attended with spasmodic constriction of these, and if coronary atheroma coexists the danger to life is enhanced. Coronary atheroma without general arteriosclerosis is manifested by dyspnœa without pain but with the signs of failing heart already described. Coronary atheroma *with* arteriosclerosis is evidenced by attacks of true angina pectoris with all its fatal augury. It follows

that the determination whether or no the arteries present signs of thickening is of the highest practical importance. The question of treatment arising out of these considerations will be dealt with hereafter.

The alliance of arteriosclerosis with *mitral stenosis*, though much less frequent than those associations already discussed, is an important one that has hitherto received but little attention. The cases of mitral stenosis observed in the early periods of life and until the age of about thirty-five are almost invariably due to rheumatic endocarditis, the forms of rheumatism manifested therewith being frequently very slight and often undetected. In later periods of life obstruction of the mitral orifice is commonly associated with arteriosclerosis. Goodhart found that in a large number of cases of chronic renal disease there was a notable and sometimes extreme thickening of the mitral flaps. In about one-fourth of the consecutive cases of chronic renal disease—192 in number—which he examined, the mitral valve was thickened or actually contracted. G. N. Pitt also noticed the association of mitral stenosis with gout and granular kidneys. He found from the records of the post-mortem department of Guy's Hospital during a period of ten years that a contracted mitral orifice is three times as common among patients with granular kidneys as in other patients. Two-thirds of all the cases of mitral stenosis occurred in women. Dr. Pitt believed that in a considerable number of these the granular degeneration of the kidneys was induced by pregnancy or some uterine trouble; and thought it most probable that the stenosis was, in the majority of instances, secondary to the kidney degeneration. The following case under my own care illustrates some interesting points in this relation.

A lady, aged 52, came under my care for dyspepsia with very slight jaundice. She manifested no history of, nor predisposition to, rheumatism. There was no sign of cardiac disturbance and the heart sounds were normal. After the lapse of several months during which there had been no symptoms other than those of occasional slight dyspepsia, I found just right of the apex a rough presystolic murmur. The urine had a specific gravity of 1.020, was slightly acid, and contained no albumin. There were no signs of gout. The evidence of mitral stenosis became very marked, the presystolic murmur was followed by a blowing systolic bruit. The urine showed none of the characters of chronic renal disease, but a sphygmogram of the radial pulse indicated by its anacrotic bend some arteriole obstruction. Ten years after my original observation, signs of implication of the nervous system became first evidenced in a slight epileptiform attack and spasm referred to the throat. Ophthalmoscopic examination showed the discs to be very pale and inclined to atrophy. Slowly signs of dementia followed, and death

took place after the case had been watched for thirteen years. At the post-mortem examination carefully made by Dr. Basil Morison, the mitral valve presented the funnel form of stenosis, the orifice admitting one finger. The substance of the valve and the chordæ tendineæ were much thickened and fibrous. At the junction of the anterior and posterior flaps of the valve on each side a small fibrous papilla was interposed. One or two patches of atheroma were to be seen on the valve. The aortic valve-segments were thickened but competent; some patches of atheroma were observed on the interior surface of the aorta. In various regions—the pleuræ, the left lung, the capsules of the spleen and liver, and the cortices of the kidney—there were evidences of a chronic fibroid proliferation. The granular changes in the kidneys themselves were not advanced but the capsules were adherent; the pleural adhesions in the left chest were very extensive, and the lung was small and fibrous; the liver was somewhat enlarged and of "nutmeg" character (not cirrhotic), its capsule was firmly fixed to the anterior wall of the abdomen and to the diaphragm by old adhesions; the spleen was fixed by its capsule to the stomach and adjacent structures by fibrous bands; the dura mater was firmly adherent to the calvarium and to the arachnoid, the pia mater showed fibroid thickening noticed especially along the lines of the blood-vessels.

In this case it appeared to me that the fibroid changes in the many and various situations were greatly disproportionate to the arterial thickening. The earliest signs were consistent with perihepatitis, the evidences of which at the autopsy were the old fibroid changes in the capsule of the liver and the firm adhesions to the surrounding structures. Then followed the signs of gradual sclerosis at the mitral orifice and the slow welding of the curtains of the valve to form the funnel-mitral. During many years there were only occasional deviations from very fair health, the subject of these changes being a lady energetic in works of benevolence. I had many opportunities of examining the urine which presented quite normal characters; there was no evidence of renal inadequacy. The signs in the lung were thought to be due to the cardiac disease. There was really a progressive and abundant sclerosis in pleura and lung. Not till ten years after my first observations did signs of paralytic dementia show themselves; these were found to be due to a very extensive sclerosis without disease of the cerebral vessels. The fibrosis of the kidney was quite a late change.

Pathology.

From a review of the extant evidence and from independent investigations which I have myself made, I am led to the following conclusions. Thickening of the walls of the arterioles may be (*a*) in

the internal coat; in such case there is a gradual obstruction of the tube of the vessel in varying degrees up to complete blocking; (*b*) in the external coat; thus the disease spreads from without inward. The muscular coat is involved, but hypertrophy of this coat is by no means an invariable feature of the disease; the fibrous tissue invades it and causes its degeneration. The intima may be more extensively thickened in some situations and the adventitia in others, frequently both external and internal coats are affected; (*c*) the process of fibrosis may arise externally to the arterioles altogether, but by its extension may involve these. The case above described is a sample of many in which there is no initial arterio-sclerosis—the abundant proliferation of fibrous tissue is in many situations. Probably some cases of granular kidney commence by a proliferating change in the fibrous tissues external to the blood-vessels, others in the blood-vessels themselves. The sclerosis observed in some cases of disease of the nervous system—disseminated cerebro-spinal sclerosis, tabes dorsalis, general paralysis of the insane—in which there is no evidence of association with diseased arteries nor with granular kidney come under this category.

It is most probable that these changes are due to a soluble poison circulating in the blood. In class (*a*) this operates directly upon the internal coat of the vessel, in (*b*) the channels are the lymphatics which are so abundant around the external coat, in (*c*) the distribution is by the lymphatics in the various fibrous tissues. It is evident that this poison may be elaborated through the agency of the syphilitic microbe. Its dependence upon gout is less surely established; there is no good evidence of any relation of intensity between the two affections, but arteritis undoubtedly can occur from gout. With some forms of renal disease its relation is close, though not in notable degree with those which affect principally the secretory portions of the kidney. It would appear that the disease affecting the heart and vessels only becomes manifested through an involvement in the disease of the fibrous elements of the kidney. Such fibrosis may be primary, as in typical forms of interstitial nephritis; secondary, as in irritations of the kidney from stone or from the extension of desquamative nephritis to the connective tissue.

The muscular coat of the larger arteries, such as the radial, is usually in over-action, though capable of relaxation from causes hereafter to be considered. This is only a natural corollary of the obstructive disease in the smaller arterioles and capillaries. The blood within the arterial tubes is in greater tension than normal, owing to the peripheral obstruction plus the increased force of the left ventricle. It is probable that the muscular coat of these arteries is gen-

erally in a state of abnormal contraction from an irritation of the vaso-motor nervous mechanism whether central or peripheral.

There is a double cause of hypertrophy of the left ventricle—the peripheral obstruction due to the disease and the resultant blocking of the arterioles and capillaries in various areas of greater or less extent, and the initiation and continuance of over-plus blood pressure within the arteries from vaso-motor stimulation. A true hypertrophy of the heart-muscle may ensue, but if its own arteries become diseased, if sclerosis spreads in its connective tissue, or if other forms of degeneration occur, enfeeblement and dilatation will follow.

While arteriosclerosis is due to a soluble and diffused poison, atheroma is the result of an insoluble, particulate material which blocks the minute arteries of the wall of the vessel (the vasa vasorum), producing the series of changes already described. The local disease, atheroma, and the more diffused disease, arteriosclerosis, often concur to produce the morbid phenomena.

Physical Diagnosis.

Examined in the way previously described for investigating atheroma, the radial artery may be found to be firm, incompressible, cylindrical, rolling under the fingers as a quill, a straw, or a tendon. The pulsation felt within the artery is not sudden but gradual; the vessel is full between the beats. The cord-like pulsating vessel is often visible. The brachial artery is also observed to be firm, tortuous, like a flexuous cord and moving laterally with each pulsation. The temporal arteries are also visible, presenting similar characters. The carotids are seen and felt to beat in the neck with a heaving pulsation. The apex of the heart forcibly lifts the chest-wall outside and below the normal position, and the observer finds on palpation a powerful thrusting impulse. Auscultation of the heart shows the aortic second sound to be loud, metallic, or tympanitic, the first sound at the apex prolonged and dull. These are the typical signs of an advanced case of arteriosclerosis, and where there is such an assemblage of signs there can be no doubt that chronic Bright's disease concurs.

The signs of thickened arteries may, however, be manifested in much less degree; so it becomes necessary to ascertain whether or no the pulse is one of high or abnormally increased tension. The method which I recommend is to place one finger, which may be the forefinger or middle finger, lengthwise over the artery, while the patient's hand is either supported by the left hand of the observer, or else by the other fingers of his right hand grasping the wrist. In this way one finger receives the impressions from the vessel over a

considerable portion of its length, and the observer should propound to himself certain questions, viz., 1. Is the pulse, with the very slightest pressure exerted, easily felt? 2. With a slight increase of the pressure does it cease to be felt? Is it readily observed with slight pressure, and easily obliterated by slight increase of the pressure? In such a case the pulse is one of low tension. Again, 3. On increasing the pressure, does the pulse become more easily felt? Is it scarcely perceptible at low pressure, while with slight increment of pressure it becomes more evident? On increasing this pressure does it cease to be thus evident? If such is the case, the pulse will be one of moderate tension. Then, 4. Does the pulse, with considerably increased pressure, become, not obliterated, but still more evident than previously, and does the artery feel hard with no very considerable change in its bulk at the time of the pulsation? If so, the pulse is one of increased and protracted tension. In certain cases, with all the pressure that can reasonably be employed, the pulse fails to be extinguished by the finger, the observer may in such case conclude that there is greatly increased tension. Then the tips of the fingers used in the ordinary way for feeling the pulse may be made to firmly compress the artery, and the flow of blood being completely arrested, it may be determined whether the vessel on the distal side of the compressed point is to be felt or not. Usually it cannot be felt, but if the coats are abnormally thick its cylindrical outline is to be detected. In some cases even, as Douglas Powell has pointed out, a pulsation is to be felt—there is a reflux pulse from an unusual inosculation between the radial and ulnar arteries, "a high tension pulse with peripheral reflux."

In a pulse manifesting these signs of over-tension of the blood current and apparent if not real thickening of the walls of the vessel, a relaxation may be brought about by various agencies. In conditions of general fever, the pulse may become compressible and dicrotic. So also after the administration of aperients, especially mercurials. Nitroglycerin and the nitrites produce a similar effect, and this is rapidly manifested after the administration of nitrite of amyl by inhalation. These are all matters of common observation. I am at a loss to understand why this should be cited as an argument against the deduction of those who assert that there is a fibroid change in the minute arteries and in favor of that of those who contend for a pure muscular hypertrophy. It has been said that the observed results give every proof of a full physiological activity in the muscular coats of the arterioles, and the latter cannot therefore have undergone fibroid change. This might be valid if it were contended that the changes were universal in the arterioles and larger

arteries, but no one supposes this. There is a consensus of modern observations to show that fibroid changes do occur in the arterioles in many situations, that these are greatly in excess of any hypertrophy of the muscular coat, and that in some parts the muscular coat is impotent from degeneration. Though there may be diseased arterioles in many areas, there is abundant effective muscle in arteries of the calibre of the radial as well as in the vast number of the smaller arteries.

Mechanical Means for Determining Arterial Resistance.—A large number of instruments have been devised for ascertaining the amount of pressure to be exerted upon the radial artery in order to arrest the blood current therein. These have been termed sphygmoscopes, sphygmometers, and sphygmomanometers. Recently Dr. George Oliver has devised instruments termed the *arteriometer* and the *pulse pressure gauge*, with which he has made many observations. The purpose of the arteriometer is to determine the calibre of the artery investigated, by noting by means of a pressure gauge provided with a dial-index the range between the point indicating the slightest degree of pressure required to make the index respond to the movement of the blood, and that which suffices to arrest the movement. Dr. Oliver finds that in health the calibre of the arteries is always varying within certain limits. A change in the calibre of the artery in health is almost invariably brought about by an alteration of posture, as from the standing to the sitting or recumbent position. "In health the calibre of the systemic arteries never remains persistently uniform, but is constantly varying within pretty wide limits in response to change of physiological condition; and the variations follow a definite order." [18] In chronic interstitial nephritis, myxœdema, acquired syphilis, and chronic gout, the calibre has been found uniform in all the postures. Dr. Oliver's observations show that the demonstration of such uniformity may have an important bearing upon diagnosis, especially in acquired cases of syphilis. In some cases, however, the detection of a uniform radial calibre may indicate a transitory condition of little or no pathological significance. Confirmatory signs must be looked for. Another valuable piece of evidence as shown by the instrument is a persistent reduction of the normal calibre of the artery. Under normal conditions the average maximum calibre of the radial artery in men varies from 2.0 to 2.5 millimetres, and in women from 1.8 to 2.3 millimetres. Organic reduction of this calibre is met with in arteriosclerosis, in syphilis, and in chronic gout. The conjunction of invariability of calibre in various postures and of reduction of calibre below the normal, is important evidence of a lasting morbid condition which leads strongly to the diagnosis of the disease mentioned.

The *sphygmograph* gives important evidence in the diagnosis of arteriosclerosis. In chronic renal disease for a protracted period, there is abnormally increased arterial tension. In taking the sphygmogram the observer finds that when slight degrees of pressure are put upon the artery the needle of the sphygmograph fails appreciably to move. The maximum movement of the needle and the satisfactory delineation of the trace are only obtained when considerable pressure is exerted. The greatest possible pressure may fail to extinguish the trace, and in some cases it may be noted that under the higher degrees of pressure the needle becomes arrested and the ascending wave broken, an anacrotic wave bulging therefrom. In other cases the first wave may be manifested as a broad plateau, forming the summit portion of the trace. The signs, therefore, besides the broad summit-plateau of prolonged tension are: (1) In taking the sphygmogram, low degrees of pressure upon the artery fail to develop the trace, while further compression succeeds; (2) the chief descending wave, the dicrotic, is but slightly pronounced, and has a higher position than in the normal trace.

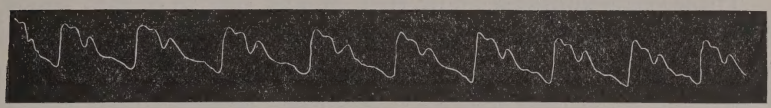
The sphygmogram in prolonged tension may have a considerable or a very slight altitude. If the pulse is full, the amplitude will be considerable; on the other hand, a pulse may be very small and narrow, and yet manifest over-tension. It is a very common mistake to say that a small thready pulse is essentially weak. The sphygmographic evidence may correct this error. In the case of a small pulse whose characters may be determined with difficulty by the finger, a hypertrophied left ventricle may be toiling against the obstruction of thickened and contracted arteries. The sphygmogram in advanced stages of chronic Bright's disease with arteriosclerosis often manifests many serrations, indicating vibrations of the needle, in all the parts of the sloping down-stroke. These are seen in extreme degree in atheroma when the ventricle begins to fail. They are recorded in Pl. III., Fig. 2, in the diastolic portion of the trace.

In the later stages of the disease also, when there is failing heart, the dicrotic wave which has previously been but slightly pronounced and near the summit of the sphygmogram becomes more pronounced and the notch is seen to be deeper. Fig. 3.

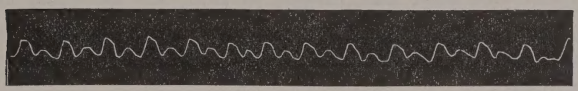
I have scarcely ever observed the conjunction of broad summit and marked dicrotism without the patient manifesting the signs of failing heart. I have noticed it when there have been concurrent signs of hypertrophy and dilatation and when there has been evidence of atheroma of the aorta. I have seen it in cases of alcoholism when degeneration of the heart might be suspected, in some instances of rapid heart and irritable heart in which commencing dilatation was

1. Chronic Renal Disease with Quick Pulse.

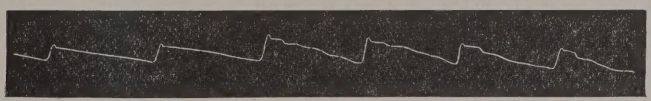
2. Central Granular Kidney. Anacrotism. Many Vibrations in Descending Curve. (After Bristowe.)



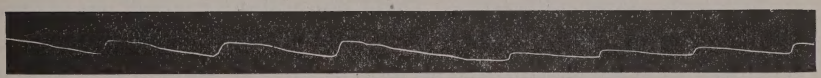
3. Contracted Granular Kidney. Marked Dicrotic Wave. (Bristowe.)



4. Pulse of Arteriosclerosis, relaxed and showing full Dicrotism after Administration of Nitroglycerin.

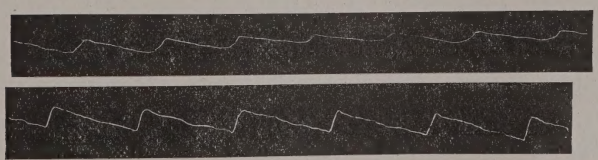


5. Typical Angina Pectoris.



6. Typical Angina Pectoris.

7. Early Signs of Chronic Renal Disease.



8. Habitually Contracted Arteries in the Subjects of Pain, Anxiety, and Precordial and Abdominal Discomfort.

PLATE III.—SPHYGMOGRAMS ILLUSTRATING CASES OF ARTERIOSCLEROSIS.

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probable, and in others manifesting hypochondriasis or melancholia. In one such case suicide was the end. It must be remembered, however, that these observations do not apply unless the dicrotism is found to be permanent. There may be many causes for a temporary reduction of tension to the extent of producing full dicrotism in the pulse of the subject of arteriosclerosis. A state of fever in the patient will produce it. So also the administration of nitroglycerin or the inhalation of nitrite of amyl. This is well seen in Fig. 4, Pl. III., where in the pulse of habitually full tension of arteriosclerosis the administration of nitroglycerin has caused full dicrotism.

These considerations show that, at any rate in certain stages of the disease in the subject of firm and thickened arteries, the muscular coat of the vessel is not closely adapted to its contents. The vibrations of the thick-walled, and in some parts atheromatous, vessel are not controlled by the contraction of the muscular walls. The ample dicrotic wave shows that the vessel is not full between the beats. The evidence is strong against the view that a true muscular hypertrophy, in arteries of the calibre of the radial, is in existence under the circumstances.

In the cases of arteriosclerosis manifesting angina pectoris, except those associated with marked incompetence of the aortic valves, the sphygmograph tells of a contracted artery whose muscular wall contracts to still greater degree during a paroxysm. There is evidence of vaso-motor spasm producing a tight constriction of the arteries against which obstruction the heart ineffectually struggles (Pl. III., Figs. 5 and 6).

Giving these considerations their due weight I would emphasize the importance of the precise investigation of the tension of the arterial pulse in cases manifesting signs of angina pectoris or of a form of suffering resembling it. If a patient complaining of symptoms which closely resemble those of angina pectoris manifest on repeated examinations, and especially on examination during the continuance of the painful crisis, low arterial tension, it is almost certain that the affection is a false, and not a true, angina. If, on the other hand, the symptoms, even though they do not seem to be typical, are attended with augmented arterial tension, the fear, present or remote, of true angina is justified. The observation is important for diagnosis, prognosis, and treatment. For diagnosis, because it suggests the probability of the occurrence of atheromatous changes in the coronary artery; for treatment because the recognition of such probability and the prolonged administration of arterial relaxants, especially the iodides (as recommended by Huchard) or nitroglycerin, may prevent the progress of the endarteritis, and may abolish the symptoms of pain.

An important indication of a contracted artery—of a condition in which the vaso-motor power causing constriction of the artery is imperfectly antagonized by a toiling left ventricle—is seen when the sphygmogram presents a sloping instead of a vertical up-stroke. When there is no indication of aneurysm or of organic disease at the aortic orifice the presence of obliquity of the up-stroke may be of considerable practical importance. First, however, the observer must be careful to ascertain whether the obliquity is occasioned by causes external to the vessel explored. The intervention of a considerable quantity of subcutaneous fat, or of any growth superficial to the vessel, may so interfere with the motion imparted to the needle of the sphygmograph as to cause a very marked obliquity of the up-stroke. Excluding such causes, as well as aneurysm and organic disease of the aorta and its valves, a sloping line of ascent observed under various gradations of pressure indicates that a feeble left ventricle is toiling to overcome the resistance of a constricted artery. We may observe such a condition of abnormal arterial constriction long before the occurrence of signs of chronic renal disease. It may be met with for long periods in patients manifesting anæmia, disappearing when the corpuscular richness returns to the normal. In painful affections—as neuralgia, migraine, or lead-colic—the sphygmograph reveals abnormally increased arterial tension (Pl. III., Fig. 8). A like character of the pulse-trace is seen in sufferers from gout, in some of the subjects of alcoholic indulgence, and in those who live not wisely but too well. So also in hysteria. In some cases the tendency would seem to be hereditary. I have found that those who manifest these signs are especially emotional and frequently ailing with painful affections. In syphilis I have found the sphygmographic evidence of over-tension in the arteries at early as well as late stages after infection. Such observations as I have made confirm Dr. Oliver's view expressed in the preceding paragraphs.

The presence of obliquity of up-stroke of the sphygmogram with prolonged tension in the artery may be of importance in diagnosis and prognosis. In cases manifesting these signs I have found symptomatic evidence of cardiac enfeeblement: precordial discomfort, faintings, extreme lassitude, inexplicable languor, accelerated breathing or dyspnœa, and nervous apprehension. In a case in which the distress of the patient seemed disproportionate to the physical signs of illness, I often found the explanation in this condition of the heart. In some cases there were alternations with palpitations or with irregularities. I also found the condition subsequent to diphtheria. In one patient right hemiplegia and aphasia ensued, another died of apoplexy. Many patients were the subjects of osteoarthritis; in other

cases emphysema, chronic bronchitis, and spasmodic asthma coexisted; in many there was marked evidence of blood deterioration.

Clinical Groups of Arteriosclerosis.

Group I. *Renal Disease.* In all forms of renal disease there may be *plus* tension in the arteries. In the earliest stages of acute nephritis the tense artery may be felt. It has been contended that this abnormally increased tension may be found previously to the commencement of any kidney change or to the appearance of any albumin in the urine. ("The Etiology of Bright's Disease and the Pre-Albuminuric Stage," by F. A. Mahomed, Medico-Chirurgical Transactions, 1874, p. 197.) In the more chronic cases of tubal nephritis the thickened arteries may be detected in some subjects, children as well as adults, with the concurrent signs of hypertrophy of the heart and retinal hemorrhages. The observation of the degree of tension in the radial artery is therefore of much importance in any case of ordinary inflammation of the kidneys. In a case which runs a favorable course the artery, when the dropsy subsides and the urine becomes less albuminous, is found to be more compressible. Conversely an unfavorable opinion must be given, if, although the acute signs subside and the albumin in the urine diminishes to a small proportion, the artery remain tense and the left ventricle shows signs of hypertrophy, and especially if in addition retinal hemorrhages are observed.

In the renal disease induced by lead-poisoning Professor Oliver, of Newcastle, finds that there is first an acute parenchymatous nephritis and afterwards a true interstitial nephritis, the walls of the small blood-vessels becoming thickened, Bowman's capsule being rendered more fibrous and laminated, and the glomeruli becoming compressed. There are, however, in lead-poisoning concurring morbid phenomena in the systemic arteries. During attacks of lead colic the pulse becomes hard, resistant, and decidedly retarded. In patients in whom there have been repeated attacks of colic, or in those who have suffered only one severe attack, there is a remarkable difference between the pulses of the two sides—nearly always there is a higher arterial tension in one radial pulse than in the other.

In cases with the symptoms of renal calculus the observation of morbidly thickened arteries is also of great importance, for in these as well there may be a like series of cardio-vascular changes.

In the majority of cases, however, of chronic Bright's disease (interstitial nephritis) with the associated arteriosclerosis there are no symptoms of acute disease and none expressive of a notable dis-

turbance of the kidney to mark the onset. Dyspepsia with the signs of gastric catarrh, headache, difficulty of breathing, or recurring bronchorrhœa may be the symptoms for which the physician's aid is sought. Then the evidences of thickened arteries with hypertrophied or dilated heart, the anæmia, the history of frequent micturition, especially at night, and the copious pale urine of low specific gravity, slightly albuminous, complete the diagnosis.

Among the factors of the morbid conditions of arteries, heart, and kidneys, mental overstrain is probably more frequent than is generally admitted. The influence of protracted emotion in the production of arterial and cardiac disease, though not often readily demonstrable, is nevertheless real. It has been proved by the plethysmograph of Mosso that emotion induces a spasmodic contraction of the arterioles. "Strong and repeated emotions," says Huchard, "can induce cardiac affections by their incessant action upon the peripheral circulation. This is one of the reasons why, according to my observations, arteriosclerosis is so frequent in members of our profession. It is the disease of medical practitioners, because their profession is associated with emotional and nervous overstrain. For the same reasons it is a disease of politicians and financiers" ("Maladies du Cœur," etc., p. 78).

Group II. *Spasmodic Asthma and Pulmonary Affections.* One of the most marked examples of firm, thick-walled, incompressible radial arteries I have ever observed was in a case in which, during many years and until the death of the patient, there was no indication of disturbance of the function of the kidneys, no albuminuria, no reduction of the normal specific gravity of the urine; but there were frequently recurring attacks of spasmodic asthma, signs of persistent local fibrosis of the lung, and occasional evidence of acute localized pneumonia.

The association between vascular emphysema of the lung and increased tension within the arteries has been noted by many observers. It is well known that chronic Bright's disease coexists in many cases. Gull and Sutton found the kidneys granular and contracted in twenty-two out of thirty-three cases in which the lungs were emphysematous.

The arteries may be found thickened in chronic pneumonia (fibroid phthisis, cirrhosis of the lung). The observation of a firm, incompressible pulse in a case manifesting signs of consolidation of the upper lobe of one lung may be very important for diagnosis and for prognosis. It tends to negative the conclusion that there is extensive and progressive tuberculosis; in such case the pulse is of low tension. It suggests that the disease is of a chronic nature and

attended with the changes of fibrosis in the lung or in the lung and pleura.

Group III. *Syphilis.* The arterial affection in syphilis occurs in two forms—the local and the general. The anatomical characters of the local forms of arteritis have been already described (p. 554). The current of recent investigation is to show that these forms frequently occur at much earlier periods after the primary infection than has been formerly supposed.

The contracted state of the arteries after syphilitic infection has already been noted. This is far more common than any proved local arterial disease. To this cause may be referred the marked anæmia of syphilitic subjects generally. The tissues are starved because of the imperfect arterial supply, and in some cases, especially in infants with congenital syphilis, œdema results.

Group IV. *Gout.* The existence of arteritis in gout has been noted by many observers. According to my own experience, however, it is not in the subjects of acute typical gout that we usually find the general signs of thickened arteries. Granular kidney is found more frequently in those who have never been the subjects of true gout. Atheroma on the other hand is much more common in these subjects.

Treatment of Arteriosclerosis.

The treatment of the conditions under which arteriosclerosis is manifested is a subject of too wide a scope to be entered upon here. The therapeutics of syphilis, gout, lead-poisoning, the various forms of pulmonary fibrosis, renal disease, and many other morbid states in which the arteriocardiac disease may be manifested, are discussed in other portions of these volumes. The principles which should guide us in our treatment of atheromatous disease of the arteries, with which a more or less widely spread arterial thickening and associated pathological changes in the heart are found in the majority of cases, I have already considered (p. 538).

I think it well, however, to make some allusion to the treatment of a prodromal state which, in my opinion, may dispose to thickening of the arteries in various situations but especially in the kidneys, because errors of treatment and of hygienic management in this stage are very likely to occur.

We will imagine that an individual who has attained, or is approaching, the age of fifty becomes fanciful, introspective, low-spirited, and moody, but shows no more obvious signs of disease than those of dyspepsia. We find that the surface and the mucous

membranes are somewhat anæmic. There is a complaint of feebleness of the heart, the subjective symptoms of heart disturbance and simulated heart failure are frequently present. A casual examination of the pulse at the wrist confirms the view that the force of the circulation is weakened. The opinion "weak circulation" is a great solace to the patient. Very probably he has made the diagnosis himself and has proceeded to fortify himself by "nips" of alcohol, by strong meat juices, or by various preparations of iron, even if he has not on his own account strengthened his heart by doses of heart tonics—digitalis, strophanthus, or cactus. But the diagnosis is erroneous and the treatment faulty. There is a false and not a true anæmia. The arterioles of the surface and of the mucous membranes are too contracted. The pulse at the wrist, which seems difficult to discover and is thus set down as feeble, is small and cordy, but can be felt on firm pressure. The indications are not for agents which increase the contractile power of the already constricted arteries, but, on the other hand, for the means which relax the arterial spasm, so as to liberate the left ventricle from the obstacle which prevents its free emptying, and thus obtain for the tissues a freer irrigation with blood.

There are strong probabilities that in such a case as this the mental depression and the subjective symptoms of pain, which are taken as the consequences, are really the most potent factors of disease. The immediate result of the disturbance of the nervous equilibrium is a vaso-motor excitation and thus a contraction of the arterioles and, from a continuance of this state, a defective arterial blood supply and a manifest anæmia. In the case of the renal arterioles this is evidenced by an abnormal diuresis, although there is a defective elimination of excretory products. In the case of the muscle arterioles there must also be a correlated disturbance, for the experiments of Ludwig and Hafiz have shown that contraction of the vessels of the surface causes an increase of blood supply to the muscles. The muscular arterioles are, however, modified by movements and by very complex interferences with their calibre. Let it be sufficient here to say that they are necessarily disturbed, and that such disturbance is expressed not only by a diminution of their physiological activities but by the production of the toxins of morbid metamorphosis.

It is only reasonable to infer that the pathogenic agent which produces fibroid change within or around the small arteries and the capillaries may be thus produced.

The first efforts in treatment should therefore be directed to the restoration of the normal arterial calibre. Primary digestion should be rendered as perfect as possible. All excesses should be avoided and

the meals must be of the simplest. Even an all-milk dietary should be prescribed for a time, or peptonized foods should be taken, or artificial digestives, such as pepsin or pancreatin, may be given with the simple ordinary meals. Let this be done and all so-called tonics be avoided. Coincidently the muscles should be placed in a state of physiological activity. The disposition to lassitude should not be encouraged. For those who cannot be persuaded to take a sufficiency of healthful muscular exercise, massage should be prescribed.

These means, however, may be insufficient. There are difficulties presented by the emotional and the mental conditions. The quasi hysterical state, the hypochondriacal manifestations, the migraine, and the vague but real sufferings are all attended with arteriole spasm. It has been noted that chronic renal disease exists in a very large proportion of the inmates of asylums for the insane. The signs of chronic interstitial nephritis were observed in seventy per cent. of the post-mortem examinations in one of the largest lunatic asylums in England (Colney Hatch). Dr. Herbert Bond, pathologist and assistant medical officer of the London County Asylum, Banstead, found that the prevalence of renal disease, as noticed at the necropsies, was nearly double in asylum experience that recorded in a great general hospital. The inference that the chief cause of the renal changes in such cases is to be found in the direct influence of over-indulgence in alcohol seems to me to be unwarranted. The mental and emotional disturbances often precede the alcohol craving and are themselves adequate to produce the renal disease.

The removal of the causes of mental disquiet may be in many cases impossible, but certain indications may be fulfilled; changes of scene and the insuring of a good air supply will do more than many medicines. The vague pains, the migraine, and some of the more acute neuralgias may be relieved by phenacetin, antipyrin, gelsemium, or quinine. Insomnia may yield to chloralamid, chloralose, sulphonal, paraldehyde, or a course of the bromides combined with small doses of arsenic. Treatment by morphine or hyoscine may be necessary in some cases, but the administration of such drugs should be not too frequent.

Thus a state of individual comfort being brought about, the mental clouds may roll away and the gradual sclerosing changes, which would otherwise eventuate in the incurable disease, may be averted.

The cautions against agents which unduly raise the blood pressure are even more necessary in cases which show signs of local atheroma as well as generally increased arterial tension, and still more when there are any evidences of angina pectoris. In these there are indications for a prolonged medicinal treatment, and the

agents should be the alkalies (the salts of potassium, which may act as depressants of the muscular fibre of the heart, being used in less degree than the other alkalies) and the iodides. I have had abundant evidence to show that a protracted course of administration of the iodide of sodium can do signal service in arteriosclerosis, especially when there are manifestations of pain of the nature of angina pectoris. "L'iodure," says Huchard, "est le médicament artériel par excellence."

The general rules for the administration of the iodides have already been given under the head of chronic aortitis (atheroma). In cases which have manifested symptoms of angina pectoris the treatment by the iodides should be prolonged. Huchard says that from fifteen to forty-five grains daily of the iodide of sodium or potassium should be administered during a period of from two to four years; and for several months after every anginoid sign has disappeared. The sodium iodide is probably less depressant, but the potassium salt is more rapid in its power of amelioration. It is well to substitute for some weeks the one for the other. That these drugs are dangerously depressing when thus administered for protracted periods is a common error. I have frequently observed an increase of the painful symptoms and of the signs of general enfeeblement when the iodide has been omitted and so-called tonics substituted. The best tonic to combine with the iodides is arsenic, which may be given in the form of Fowler's solution in three- to five-minim doses three times a day after meals. I have had abundant evidence that the continuous administration of the iodides mitigates and tends to abolish the painful manifestations. These become less and less and in some instances cease altogether. I am prepared to admit that in a certain proportion of cases, provided the treatment be commenced sufficiently early, there is a complete restoration. On the other hand, though the crises of pain may be abolished by the treatment, death may take place from cardiac failure. This is not to be wondered at when one considers the evidence derived from morbid anatomy. If the changes in and about the coats of the vessel are capable of removal by absorption, or if the constriction of the walls, however produced, is capable of such relaxation as to restore a sufficient circulation, the morbid symptoms may be removed; but if, as from calcareous transformation, the obstruction be incapable of removal, even though the pain be abolished (and this in itself is a considerable gain, for every attack of pain is a heart-shock), the heart muscle must in time inevitably fail. If there are evidences of intolerance of the iodides in a case manifesting angina pectoris, then (*a*) the tincture of iodine may be administered in five- to ten-drop doses in half a wineglassful of claret or marsala,

during meals, three times a day, or (*b*) five minims of the iodide of ethyl (preferably from the glass capsules) may be inhaled from four to six times a day; (*c*) in certain cases or for certain periods, instead of the iodides, trinitrin or nitrite of amyl may be prescribed, as previously stated. Methylal and pyridine have been used to prolong the good effect of the inhalation of nitrite of amyl in angina pectoris. Or the patient may be instructed at the onset of an attack to swallow a tablet containing $\frac{1}{100}$ grain of nitroglycerin and also immediately to inhale the contents of a capsule of nitrite of amyl. It is seldom that these means do not calm or avert an attack of angina pectoris, but when the pain is of extreme intensity the hypodermic injection of hydrochlorate of morphine gr. $\frac{1}{4}$ or $\frac{1}{3}$, or the administration of opium may be necessary.

I have said that as a general rule and for the systematic habitual treatment of cases manifesting arteriosclerosis, as well as for the symptomatic therapeutics of attacks of angina pectoris, the administration of digitalis and other forms of cardiac tonics is not to be recommended. This prohibition must not, however, be absolute. It must be remembered that at certain times and under certain conditions in a severe case the muscle of the heart may be gravely weakened. This enfeeblement may occur after an attack of angina pectoris as a result of the shock of the pain. It may occur also in the form of syncopal attacks in the absence of pain. At such times of imminent heart failure I know of no agent that acts more promptly for reanimation than *musk*, which can be placed in powder upon the tongue and swallowed down with a teaspoonful of milk; or repeated hypodermic injections may be practised of (*a*) pure ether and brandy equal parts, (*b*) a saturated olive oil-solution of camphor, or (*c*) nitrate or sulphate of strychnine ($\frac{1}{50}$ to $\frac{1}{20}$ grain). With either of these hypodermic solutions may be combined $\frac{1}{50}$ grain of digitalin. If digitalin be thus administered no further dose should be given till after the lapse of twenty-four hours. If digitalin has not been thus given hypodermatically, digitalis or digitalin may be administered by the mouth according to the rules already given.

There are certain symptoms which call for a more protracted administration of digitalis or other cardiac tonic in cases of arteriosclerosis; these are: (*a*) Dilatation of the heart chambers without valvular imperfection; this is evidenced by enlargement of the area of cardiac dulness, displacement of the apex outward or downward, heaving pulsation of the right ventricle below the ensiform cartilage, and increasing dyspnœa, especially on exertion; (*b*) The manifestation of a systolic murmur and other signs indicating regurgitation from the ventricles; when mitral regurgitation is thus the sequel of over-

pressure in the aorta the typical attacks of angina pectoris usually cease, though the distress of dyspnœa may increase; (*c*) The physical signs of dilatation of the heart and of regurgitation at the orifices may be wanting, but there are physical evidences of œdema of the bases of the lungs and perhaps some general dropsy. In such cases digitalis or other cardiac tonic is called for, but it is best that it should be administered in combination with trinitrin or the iodides. Dr. G. W. Balfour says: "Digitalis cannot be safely given to senile hearts without simultaneously unlocking the arterioles." I am of the same opinion. Strophanthus is more uncertain in its action, but is valuable in some cases. Convallaria majalis has also, according to my own observation, proved very useful. I have seen cases in which, under its influence, the dyspnœa and dropsy have disappeared coincidently with free diuresis.

It is well, when such signs of failing ventricles as I have mentioned are manifested, to commence with digitalis (half an ounce of the infusion, ten minims of the tincture, or one grain of the powdered leaves) and combine with one-drop doses of the solution of nitroglycerin (1 to 100) or the tabloids containing $\frac{1}{100}$ grain, the doses to be administered three times in the twenty-four hours; or instead of the preparations of digitalis one granule of Nativelle's crystallized digitalin may be given once or twice in the day for a short period of days. The nitroglycerin should still be administered thrice daily, but after a time the dose may be decreased to $\frac{1}{200}$ grain. If in the course of three or four days there are no signs of improvement, a combination of cardiac tonics should be tried—digitalis with convallaria or strophanthus, or occasionally the last two without the digitalis. Nux vomica or strychnine may be administered with great advantage in combination with these. After one or two weeks of the administration of the nitroglycerin, the iodides in combination with the cardiac tonics should be substituted, and these should be continued, with occasional substitution of the nitroglycerin, for protracted periods. In cases where the chief failure is in the right ventricle, where venous turgescence or venous pulsation is manifested, the too much neglected venesection from the arm is a most valuable measure. Cardiac tonics are powerless for good when the right ventricle is overdistended or when its every systole forces a considerable back wave into the venous system. After a bleeding of six or eight ounces from the arm the distention may be relieved, and the cardiac tonics, which were powerless before, then become effectual. I have seen many cases in which the lesson of the value of venesection in these cases has been taught. One such abstraction of blood often relieves, but it is usually necessary to repeat the operation after the lapse of a few days.

Aneurysms of the Systemic Arteries.

Aneurysms of the aorta constitute nearly half the total of aneurysms observed, and of these the great majority are in the thoracic portion. The relative prevalence of aneurysms in the systemic arteries of the body, according to the tables of Crisp, may be thus expressed, the numbers indicating the proportions in a total of 551 cases in English records and 364 specimens in London museums: I. Popliteal artery (187 instances). II. Femoral (78). III. Subclavian (35). IV. Carotid (34). V. Axillary (26). VI. Innominate (23). VII. External iliac (16). VIII. Common iliac, posterior tibial (4). IX. Gluteal (2). X. Internal iliac, temporal, vertebral, subscapular, brachial, and radial (1).

Morbid Anatomy, Causes, and Modes of Development.

For the development of aneurysm in a systemic artery there must be a local weakening of a limited portion of the arterial wall. The conditions of disease which dispose to this local softening are similar to those already discussed in the section on the etiology of aneurysm of the aorta—there must be a subacute or chronic arteritis in the one case as there must be a subacute or chronic aortitis in the other. The predisposing causes are the same in both cases. I cannot doubt that syphilis is a potent cause, for it is not uncommon to find multiple aneurysms in syphilitic subjects. Gout, alcoholism, and the other causes of atheroma are also factors.

In the case of aneurysms of the systemic arteries direct overstrain is a more pronounced cause than it is in the case of aneurysm of the aorta. It is shown that the systemic artery most liable to aneurysm is the popliteal. This vessel is especially liable to injury from sudden flexing of the leg. As it emerges from the popliteal space it lies between the two heads of the gastrocnemius, and forcible contraction of this muscle tends to constrict it. It is clearly shown that popliteal aneurysm is frequent in those persons whose occupations cause them to make severe muscular efforts while in a standing position. The immediate effects are an increase of blood pressure within the vessel and a local compressing violence to the portion of the arterial wall within the grip of the contracting muscles. It is most probable that the initial morbid change is in the nutrient vessels of the arterial wall, the vasa vasorum. An aneurysm may result from a wound in an artery which has cicatrized but has yielded to the internal pressure of the blood current.

Another cause which operates in the case of the systemic arteries,

although inappreciably in the case of the aorta, is *embolism.* A clot or vegetation detached from a heart valve, from any part of the lining membrane of the aorta, or from one of the larger arteries, may be carried in the blood current and become fixed against the intima of an artery of smaller calibre. There it acts as a local irritant causing softening of the arterial wall, and the latter becomes dilated by the force of the blood current. The dilatation is fusiform when the whole canal of the artery is thus plugged, saccular when the clot adheres to one side of the tube. The cerebral arteries are those which are most frequently affected in this manner, but the brachials or the branches springing therefrom or the femorals may suffer. The embolus is usually arrested at the bifurcation of an artery, but a secondary thrombus may form in the distal part of the branch which is blocked. The resulting arteritis, the consequent dilatation of the vessel, and the rupture into the surrounding tissues are all usually (except in the case of the cerebral arteries) attended with much pain. The sequence of events may be diagnosed when such severe local pain with throbbing of the artery above and loss of pulsation below are observed in a case which presents signs of valvular disease of the heart or infective endocarditis. The infective agencies may be such as to induce pus formation—an abscess may form especially in situations where, as in the axilla, there is much loose cellular tissue. There may be sloughing of the sac, or the inflammation may extend to the healthy portions of the artery and set up a plastic arteritis. In very rare cases this plastic arteritis has been curative; it effectually sealed the vessel, and when the sac became separated the aneurysm was cured. If, however, the inflammation be not plastic but suppurative, the arterial wall becomes softened and inflamed, and hemorrhage, probably fatal, occurs.

In the early stages of aneurysm the sac is constituted by all the coats of the artery. As the bulging increases the middle coat (afterwards the internal coat) disappears and then the sac is formed by the thickened external coat only. New growth of fibrous tissue takes place and the normal structure of the external coat is lost. As the enlarging aneurysm presses upon surrounding structures, these become necrosed, and inflammatory changes also supervene coincident with this necrosis; bones become eroded, muscles and fibrous tissues are matted together; and these altered structures become incorporated with the sac constituted by the dilated external coat of the vessel. Ultimately the sac may be constituted only by the welded-together tissues outside the arterial coats, and, the pressure causing more and more necrosis and thinning of the cutaneous tissues, may burst externally.

In the great majority of aneurysms coagulation of the contained blood takes place during life so that there are produced stratified layers of fibrin lining the sac. The coagulation first takes place upon the morbidly changed lining membrane; the rapid movement of the blood tends to carry away, to "whip out" as it were, the red blood corpuscles; the blood pressure compresses the successively formed layers of coagula against the wall of the sac. Any cause which retards the passage of blood through the diseased vessel retains the red blood corpuscles, which find their way as red streaks or red coagula between the layers of whitish fibrin. The formation of coagula of fibrin favorably strengthens the sac and arrests the force which tends to make it progressively dilate. The coagula may so increase as to block the orifice in the healthy artery from which the aneurysmal dilatation springs. Or the enlarging sac may press upon the healthy artery on the cardiac side and may thus diminish or obliterate its calibre. Thus in very rare cases the aneurysm is self-cured. The coagula may completely fill the cavity. In such case, of course, the pulsation in the sac ceases; in course of time the firm tumor shrinks while the anastomotic vessels enlarge, and the circulation in distal parts is by their agency restored.

Aneurysms of Arteries of the Lower Extremities.

An aneurysm in the *inguinal region* may have its origin in the external iliac or in the femoral artery. Its origin and course may be marked by no special symptoms, so that it remains long unobserved until it is found as a large tumor in the iliac fossa. It may present two bulgings, one above and the other below Poupart's ligament; usually the upper swelling is the more prominent, the fascia in the thigh compressing the sac and the vessel in the latter position, while in the former there is no impediment to the expansion of the sac. Pressure of the sac on the neighboring veins (the femoral and the internal saphena) causes venous fulness and œdema of the limb below, and pain along the front of the thigh and the inner part of the knee and ankle may be caused by irritation of the genito-crural or anterior crural nerve.

Diagnosis must be made from enlarged glands, pulsating tumors, and abscesses. Care must be taken especially to differentiate this condition from abscess, for serious mistakes have been committed, aneurysms having been punctured in the expectation of giving exit to pus. In a doubtful case the patient should be anæsthetized so that the abdominal muscles are completely relaxed; the sac may then be grasped and its expansile pulsation felt.

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The *constitutional treatment* for such aneurysm should be conducted according to the lines laid down for treatment of aneurysms of the aorta, the indications being (*a*) to lessen the force of the circulation within the sac by complete repose; (*b*) to reduce the total quantity of circulating blood by restricted diet and abstinence from fluids; (*c*) to modify the vascular conditions by the medicinal administration of the iodides. If after due trial these means are found ineffectual to arrest the progress of the aneurysm, mechanical compression of the arteries, proximal or distal or both, may be tried. Proximal compression of the lower part of the abdominal aorta by a tourniquet or by the digital method, the patient being under an anæsthetic, may be made. Distal compression of the femoral artery may in like manner be practised. If the aneurysm be low down in the groin, the surgeon may ligate the external iliac artery, this operation having proved very successful; or the common iliac artery may be tied.* Ligature of the abdominal aorta and galvano-puncture have been tried, but no successful result in either case has been recorded.

Aneurysms of the *gluteal and sciatic* arteries are seated deeply in the buttock. The symptoms attending them are pain in the direction of the great sciatic nerve and impediment to the free movement of the lower limb. There may be great difficulties in the diagnosis of this affection from abscess, and it may be necessary to use an exploring needle to determine between pus and blood. In case of an aneurysm rest and constitutional treatment must be advised. Ligature of the internal iliac artery has been successful.

Aneurysm of the *femoral artery* is usually felt as a globular tumor in the upper part of the thigh, the aneurysmal dilatation arising in the portion of the artery which is superficial in Scarpa's triangle before the vessel enters the firmly fibrous sheath (Hunter's canal).

* The following table, which is compiled from the data in the elaborate work of Ballance and Edwards ("A Treatise on the Ligature of the Great Arteries in Continuity with Observations on the Nature, Progress, and Treatment of Aneurysm," London, Macmillan & Co., 1891), shows the mortality after ligature of the main arteries for aneurysm.

Artery.	Number of cases recorded.	Mortality per cent.
Abdominal aorta	7	100
Right subclavian (*first part*)	14	100
Innominate	17	94
Common iliac	55	75
Subclavian (*third part*)	251	53
Common femoral	31	51
Common carotid	789	40
External iliac	141	22
Superficial femoral	277	19

An aneurysm within Hunter's canal is rare. Aneurysm of the deep femoral artery has been caused by emboli in cases of endocarditis. In a case recorded by Sir Dyce Duckworth (*British Medical Journal*, June 14th, 1890, p. 1,356) there was sudden agonizing pain in the left knee, and afterwards, in increasing degrees, along the inner part of the thigh; there was probably irritation of the anterior crural and long saphenous nerves. A similar case of aneurysm of the deep femoral from embolus is recorded in the catalogue of the London Hospital Museum, p. 348. The bursting of such an aneurysm will be indicated by intense pain with signs of syncope.

In the case of a small femoral aneurysm, the *treatment* by flexion of the thigh may be tried. The leg and thigh should be first bandaged from the toes upward, and then the thigh flexed and fixed in this position by strap and buckle with additional support by sandbags and pillows. In twelve hours the flexion may be lessened and observation made as to whether coagulation and consolidation have taken place within the aneurysmal sac. The flexion should then be repeated. Even when there is good evidence of coagulation partial flexion should be repeated. Moderate doses of opium should be administered to the patient during the continuance of the mechanical treatment.

Instead of this plan or even in addition, Reid's treatment by the india-rubber (Esmarch's) bandage may be practised. Bandages should be applied firmly from the toes upward as far as the aneurysmal tumor; the turns should be applied more lightly over the sac of the aneurysm and continued for a short distance above. The bandages should be left on for an hour and a half. Before their removal digital compression of the artery above the aneurysm should be practised. The patient should be anæsthetized during the treatment.

The surgical method of treatment of femoral aneurysm should be, if possible, digital compression of the common femoral above the sac. Here the artery is very superficial, separated from the margin of the acetabulum and front of the head of the femur only by the psoas muscle. The vessel is readily compressed by the thumb or by mechanical means. Lower down, the artery may be compressed against the shaft of the femur. Digital compression made with the thumb, and maintained by relays of surgeons or assistants, has been effectual in several cases. The object of digital compression of the healthy portion of the artery on the heart side of the aneurysm is (by an arrest of the blood flow) to produce coagulation within the sac. It must be remembered, however, that the clot thus produced is a *soft* clot, and not like the gradually and evenly deposited layers of fibrin which are observed in the process of spontaneous cure. Yet

there may be in some cases a disposition induced by this treatment to the commencement of the process of gradual clotting. In the case of flexion of the limb or mechanical compression by a tourniquet there is an obstruction to, but not necessarily an arrest of, the blood flow—the tendency to the deposition of fibrin in layers is rendered more probable.

In the case of failure of these measures of digital, postural, or mechanical compression of the vessel, ligature of the external iliac artery is generally preferred by surgeons to that of the femoral artery itself.

Aneurysm of the *popliteal artery* is sometimes indicated at the time of its origin by a sudden pain caused by injury from muscular overstrain to the coats of the vessel. More often it arises and progresses insidiously. When arising from the back of the vessel it compresses the popliteal vein, causing venous congestion below the point of compression and subsequently œdema. It also exercises pressure upon the internal popliteal nerve and causes shooting pains down the leg to the toes with cramps and weakness or paralysis of the muscles. When the sac arises from the front of the artery it is of small dimensions; it tends to cause erosion of the femur or of the tibia, and arthritis of the knee with effusion into the joint. There is constant pain, aching, gnawing, or burning, in the joint together with stiffness. In some cases a thrill may be felt over the aneurysm, and a murmur may be heard with the stethoscope at the time of the cardiac ventricular systole.

The first symptoms of popliteal aneurysm may be mistaken for rheumatism. There is pain referred to the knee-joint; or the suffering may be down the leg to the foot, and the knee may be swollen, stiff, and painful. The swelling due to aneurysm must be diagnosed from abscess, solid tumor, pulsating sarcoma, and bursal cyst. The diagnosis of aneurysm may be made by observing that the tumor presents an expansile pulsation; that the pulsation is decreased if the limb be raised while the patient is recumbent, and that digital compression of the femoral artery causes arrest of the pulsation in the sac and perhaps a shrinkage of the tumor. By the pressure of the hand over its situation the sac may be further emptied, but it soon refills if the pressure be removed, the compression of the femoral taken away, and the leg depressed.

Popliteal aneurysm may rupture among the muscles of the calf, may extend upward and burst into Hunter's canal, or may press toward the surface and rupture through the skin. The rupture amid the muscle may be rapid or gradual. In the former case there is sudden pain and the patient becomes faint, pale, and bedewed with

a cold sweat (syncope). In the latter there are similar signs, but of less intensity; the veins of the limb may be observed to be distended and the pitting of œdema may be seen; the patient finds the limb to be numb and heavy. These are the signs of diffused aneurysm. There may be repetitions of the symptoms at intervals coincident with new hemorrhages, or these may take place slowly as in leaking aneurysms.

In the *treatment* of ordinary popliteal aneurysm, rest and the adoption of the general dietetic and medicinal means already indicated may result in consolidation of the sac and cure. This failing, flexion at the knee or the india-rubber bandage, or both measures, may be systematically tried. Digital or mechanical compression of the femoral artery has resulted in cure in a considerable number of cases. If the signs continue to be adverse, the surgeon will probably choose to tie the femoral artery; this will certainly be the course if there be any sign of rupture of the aneurysm. Some surgeons (Trélat, Peyro, and others) advocate complete extirpation of the sac, as well as ligature of the artery, and many prefer ligation of arteries to the milder measures of compression. If gangrene of the leg and foot results, the only course is amputation of the limb.

Aneurysms of Arteries of the Upper Extremities.

Aneurysm of the *innominate artery* first shows itself behind and above the inner portion of the right clavicle and may be felt between the two heads of the sterno-mastoid muscle. It may cause bulging of the sternum, the inner portion of the clavicle, and the first rib, or may extend upward in front of the carotid artery or backward toward the trachea and spine. There may be pouches extending in various directions. No projection may be visible and the outline may be detected only by dulness on percussion. The aortic second sound is loud in proportion to the fluid content of the sac. The pulse in the right radial may be modified, presenting marked differences from the normal pulse in the left radial. There may be dyspnœa, stridor, and voice impairment from pressure on the trachea and from involvement of the right recurrent laryngeal nerve. Dysphagia may result from pressure against the œsophagus. Venous congestion and blueness with œdema of the right hand and arm and of the right side of the face, head, and neck may result from pressure upon the innominate vein. Pain down the arm and shooting pains in the head and neck may occur from the pressure of the sac causing irritation of the superficial cervical plexus. Involvement of the sympathetic nerve may cause dilatation and throbbing of the

arteries of the right side of the head and face and sweating in these situations. The aneurysm may burst externally or into the trachea, œsophagus, or lung. It is to be remembered that innominate aneurysm often coexists with aneurysm of the aorta.

The *treatment* of innominate aneurysm should consist, first, in the usual constitutional means, and perhaps digital compression of the carotid artery. In case these measures fail, the common carotid and subclavian arteries should be ligated. In such case the surgeon puts in force the operation known as Wardrop's—the distal ligature. Wardrop in 1827, in a case of aneurysm of the innominate artery, tied first the carotid and subsequently the subclavian. Brasdor's operation, suggested before Wardrop's but not carried out, consists in tying a single artery on the distal side of an aneurysm, as, for instance, ligation of the upper end of the carotid artery for an aneurysm near the origin of the vessel. According to Mr. Christopher Heath, (*Lancet*, February 1st, 1890, p. 229) the ligation of an artery on the distal side of an aneurysm produces clotting by altering the direction of the current of blood.

Aneurysm of the *common carotid* artery arises usually at the bifurcation of the trunk or at the origin of the right artery. The pulsating tumor is readily detected and may attain a large size in the neck. The pressure-signs may be evidenced by dyspnœa and spasmodic cough, by aphonia, by dysphagia, by tinnitus aurium, impairment of vision, vertigo, stupor, pain and tenderness of the scalp, besides the distressing sense of pulsation. There may be compression of the larynx, trachea, pharynx, œsophagus, jugular vein, or recurrent laryngeal nerve. The sac may rupture into the trachea, pharynx, or œsophagus. A carotid aneurysm projects beneath the inner head of the sterno-mastoid and extends up the neck in the course of this muscle.

Diagnosis from a tumor of the thyroid gland is readily made, because a thyroid swelling rises and falls in the act of swallowing while the aneurysmal tumor remains *in situ*. Gland tumors of the neck are movable over the vessel. Abscess is to be distinguished by the signs of fluctuation, the absence of the distinctive aneurysmal pulsation, and the fact that compression of the artery below has no effect upon the swelling.

In the *treatment* of carotid aneurysm digital compression of the artery below the sac has been successful. The surgeon may elect to tie the artery above or below the aneurysm—the proximal ligature is to be preferred. Certain dangers, however, attend the operation. There may be syncope from the sudden interference with the normal cerebral circulation; convulsions and hemiplegia may ensue from the

resulting cerebral softening. Pulmonary congestion and inflammation also may occur, probably from the interference with the due nutrition of the vagus and its roots.

Aneurysm of the *internal carotid* artery tends to project into the pharynx; it may be mistaken for abscess of the tonsil. The finger must be used to detect its pulsation and the suppression of its pulsation when the carotid is compressed in the neck. The treatment must be by digital compression or ligation of the common carotid artery.

Aneurysm of the *external carotid* artery may cause paralysis of the side of the tongue owing to pressure of the sac upon the hypoglossal nerve. The treatment should be by digital compression of the common carotid, or, that failing, by ligature of the external carotid artery, if possible, if not of the common carotid.

Aneurysm of the *subclavian artery* is much more common in the vessel of the right side; it may spring from any portion thereof, but aneurysmal dilatation has not been observed in the first portion of the left artery. The swelling usually projects above the clavicle between the sterno-mastoid and the trapezius muscles; it may grow downward within the thorax and cause thickening of the pleura and compression of the lung. The external jugular vein may be observed to be distended. There may be œdema of the arm and even gangrene. The brachial plexus may be pressed upon or involved in the sac, the symptoms being pain down the arm with numbness and muscular enfeeblement. The phrenic nerve also may be involved.

In the *treatment*, protracted rest with the usual dietetic and medicinal measures should be tried, for a cure is by no means infrequent. The surgical treatment of subclavian aneurysm is very unsatisfactory. Simultaneous ligature of the subclavian artery immediately above the clavicle and of the common carotid artery has been successfully practised by Dr. Monod. Professor Le Denbu has stated (*The Medical Week*, February 1st, 1895, p. 56) that good results have followed peripheral ligature of the large vessels of the neck in some cases.

Aneurysm of the *axillary artery* may form on any part of the vessel. Its most frequent manifestation is as a projection below the clavicle and above the pectoralis major muscle; it may extend behind and above the clavicle; the outer portion of the bone may be forced upward. Pressing on the axillary vein, it may cause venous congestion and œdema of the hand and arm and of the adjacent side of the chest. Pain, numbness, and muscular paralysis may result from the involvement of the brachial plexus. The aneurysm may cause erosion of the humerus or of the shoulder joint into which rupture may take place, or it may penetrate into the thorax causing thickening of the pleura and compression of the lung.

Axillary aneurysm may be treated, when this measure is possible, by digital compression of the subclavian artery as it lies upon the first rib, pressure being made from above the clavicle. Direct compression of the sac may be made coincidentally. When the artery cannot thus be reached for compression, the Esmarch's bandage may be applied. If these means fail, the subclavian artery should be tied; the operation, however, is difficult and dangerous. In the case of a large aneurysm it has been recommended that the subclavian artery should be first tied and then amputation performed at the shoulder joint. Such amputation is the only course when there is diffuse aneurysm or gangrene of the limb.

Aneurysms below the axilla when not traumatic are nearly always due to embolism. In cases of aneurysm in the forearm, systematic flexion of the arm at the elbow and the application of an Esmarch's bandage may be tried. If these means fail, the ligature of the brachial artery must be performed, unless the aneurysm be superficial in the lower part of the forearm, when ligatures can be applied both above and below the sac.

Aneurysms of the Cerebral Arteries.

Aneurysmal dilatations occur not only in the larger intracranial arteries but also in the minute arteries in the substance of the brain (miliary aneurysms).

Aneurysms of the larger intracranial arteries, according to the statistics of Lebert, Durand, Bartholow, and others (as collated by Gowers), have been observed in the following relative frequency: Middle cerebral artery, 44 cases; basilar, 41; internal carotid, 23; anterior cerebral, 14; posterior communicating, 8; anterior communicating, 8; vertebral, 7; posterior cerebral, 6; inferior cerebellar, 3.

Morbid Anatomy, Causes, etc.—Aneurysms of the arteries named are in most cases saccular, rarely fusiform. They vary in size from a small pea to a large nut, but they may attain to the size of a pigeon's egg or even a hen's egg. Rupture takes place in more than half the cases, the blood escaping most frequently into the membranes at the base of the brain. The rupture is generally by a small opening, and therefore the escape of blood is gradual and, in some cases, intermittent. The hemorrhage may be into the brain itself or into the lateral ventricle through ploughing up of the brain substance. The point of rupture may be difficult to discover after death, for the aneurysm is in the midst of a coagulum; it may be found by washing away the clot slowly by a gentle stream of water.

The causes are similar to those of aneurysms of the systemic

arteries already discussed. Men are affected more than women in the proportion of three to two. These aneurysms are much more frequent before middle age than are aneurysms elsewhere. They are more common on the left side than on the right in the proportion of four to three. Embolism is the most frequent cause in the cases occurring before middle age. In these cases heart disease is usually to be discovered, or at any rate there is evidence that endocarditis or some cardiac affection has been previously manifested. In fatal cases embolisms of other organs are frequently demonstrated; in some cases aneurysmal dilatations of several arteries have been discovered, and the inflammation and yielding of the arterial coats from the impacted emboli have been traced. Cerebral aneurysms due to such cause have been found in children. The middle cerebral artery which is most prone to embolism is also the artery most frequently affected by aneurysm. Syphilis is also an undoubted cause of intracranial aneurysms. Such aneurysms have been frequently found in cases of constitutional syphilis in young persons in whom no other cause was discoverable. Gowers considers it probable that disease of the arteries due to *inherited* syphilis may cause aneurysm, as it may certainly cause cerebral hemorrhage. Chronic endarteritis (atheroma) from the causes already discussed—with which acute and subacute changes, as I have previously stated, may be associated—is a cause of intracranial aneurysm in the later periods of life. "Often there is extensive fatty and calcareous degeneration of the vessels at the base, and on one of them a distinct aneurysm" (Gowers). Injury is a rare cause, but a blow or fall on the head may induce disease of the wall of the artery owing to the extension of inflammation from the contiguous structures.

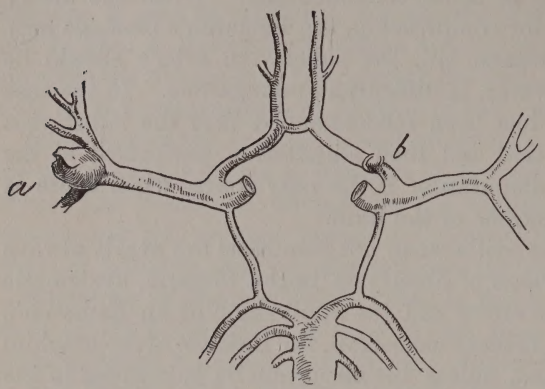

FIG. 37.—Aneurysms (*a*) at the bifurcation of the middle cerebral artery (showing the aperture from which fatal hemorrhage occurred); (*b*) near the origin of the anterior cerebral artery. (Coats.)

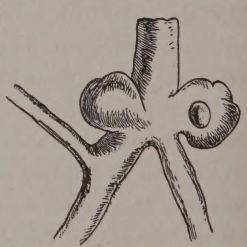

FIG. 38.—Two Aneurysms of the Left Middle Cerebral Artery; one has ruptured by a circular opening. (Sir W. Gull.)

Aneurysm of the internal carotid artery produces symptoms chiefly by compressing the optic nerve on the inner side and the nerves on the wall of the cavernous sinus on the outer side. The sight fails in the eye on the same side as the aneurysm. The third nerve also suffers early, so that ptosis occurs and is followed by paralysis of the muscles of the eyeball. There may be loss of sensibility in the globe from involvement of the ophthalmic branch of the fifth nerve. A large aneurysm may cause hemiplegia. In some cases the subject of aneurysm of the internal carotid has been conscious of a murmur, and observers have heard this systolic murmur on auscultation of the skull; the bruit has ceased when the carotid artery has been compressed. Headache and giddiness are also symptoms of this as of other intracranial aneurysms.

Aneurysm of the *basilar artery* occurs for the most part near its anterior extremity; in some cases there is a general dilatation of the whole vessel (see Fig. 36). In about one-third of the cases there have been no symptoms (Lebert). Headache may be severe; it is characteristically occipital, and giddiness is more pronounced than in aneurysms elsewhere. There may be paralysis of the limbs on one side or on both sides. The fifth nerve may suffer. In many cases no diagnosis is possible, for symptoms have not betrayed the condition until the terminal apoplexy which attends the rupture of the aneurysm. Sometimes apoplexy is caused without rupture, from the softening occasioned by the pressure of the sac.

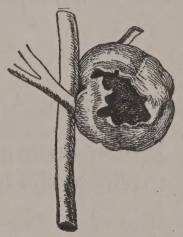

FIG. 39.—Aneurysm of the Left Middle Cerebral Artery in a Girl, Seventeen Years of Age, the Subject of Rheumatism. Some granulations were found on the mitral valve. The aneurysm was probably caused by embolism. (Sir W. Gull.)

Aneurysm of the *middle cerebral artery* occurs most frequently in the portion within the fissure of Sylvius, but sometimes it is near the commencement of the vessel and sometimes in one of the branches. Hemiplegia and convulsions are the symptoms produced in many cases. Speech is occasionally affected when the aneurysm is located in the artery of the left side, and a rupture of the aneurysm occurs in the majority of cases. Headache and giddiness followed by convulsions and partial hemiplegia are symptoms which should suggest the probability of aneurysm of the middle cerebral artery, though a precise diagnosis would be difficult or impossible.

The symptoms caused by aneurysms of the *posterior cerebral* and of the *cerebellar arteries* are too vague to be of practical value.

Treatment.—The treatment, if intracranial aneurysm be suspected, should be rest and careful diet with the medicinal administra-

tion of the iodides for a protracted period. In the case of a woman, aged forty, who suffered from right frontal headache, photophobia, impaired vision in the right eye, and paralysis of the external rectus, a systolic murmur could be heard loudest at the right temple. Iodide of potassium was given in doses increasing to thirty-six grains daily. Four months later, after a violent attack of vomiting and purging, the murmur ceased and was never afterwards heard. In five weeks the external rectus was no longer paralyzed. In this case there can be no doubt that an aneurysm existed—probably in the internal carotid—and that from a happy combination of circumstances coagulation had occurred in the sac (case by Dr. Humble, of Cerfe Castle, quoted by Gowers, "Diseases of the Nervous System," Vol. II., p. 504). Ligature of the common carotid has been successful in some cases of aneurysm of the internal carotid. It has been suggested that the vertebrals might be tied for basilar aneurysm. Dr. Alexander, of Liverpool, has shown that this operation is practicable.

Miliary Aneurysms.

(Minute Aneurysms of the Arteries within the Brain.)

It has been shown by Charcot and Bouchard that in cerebral hemorrhage the initial cause is to be found in aneurysmal dilatations of the cerebral arterioles, the rupture of which occasions the outpour of blood.

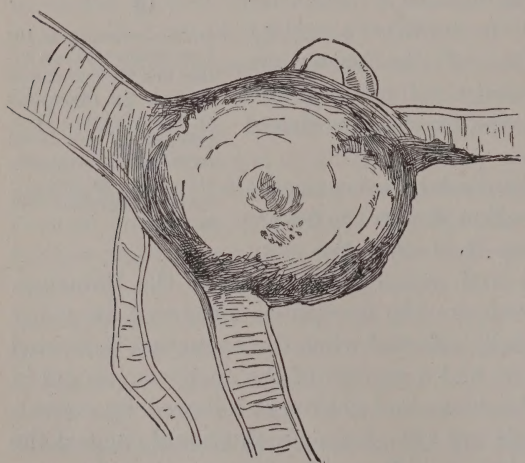

Fig. 40.—Sacculated Miliary Aneurysm of a Nutrient Artery of the Brain. × 27. The aneurysm was about $\frac{1}{20}$ inch in diameter. (Coats.)

Morbid Anatomy.—Miliary aneurysms in the brain are to be discovered at the post-mortem examination by washing away the coagula and the cerebral substance and carefully picking out the thread-like vessels. On the sides of the arteries are seen little globular bodies which are really minute aneurysms varying in size from $\frac{1}{100}$ inch to $\frac{1}{25}$ inch. (Fig. 41, *aa*.) In these the middle coat has disappeared, the inner coat having become blended with the external coat. The portions of the brain in which those were

found by Charcot and Bouchard were: central ganglia, cortex, pons, cerebellum, centrum ovale, middle cerebral peduncle, crus cerebri, and medulla oblongata—the order in which these are mentioned giving the relative frequency. The external coat of the minute aneurysmal sac is found on microscopical examination to be infiltrated with numerous round cells, which are clustered in groups; there is, as Charcot and Bouchard pointed out, a periarteritis. Other observers have stated that the process may commence in some cases by disease in the internal coat; indeed the whole of the wall of the vessel may be affected by the inflammatory process. The dilatations are frequently seen at the bifurcation of the artery or around the commencing portions of its smaller branches. The most frequent sites of ruptured miliary aneurysms are the branches which arise at right angles from the middle cerebral artery in the fissure of Sylvius and pass upward through the lenticular nucleus and the internal capsule to the caudate nucleus and optic thalamus. The vessel termed by Charcot "the artery of cerebral hemorrhage" is that branch which passes between the outer part of the lenticular nucleus and the external capsule through the former to the internal capsule. Charlewood Turner has shown that some of these miliary aneurysms may become obsolete—the contents may be coagulated, the internal coat being thickened and blended with the coagulum within it, and the external coat manifesting a large overplus of fibrous tissue.

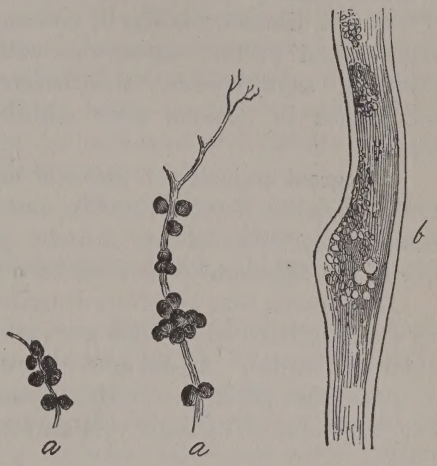

FIG. 41.—Miliary Aneurysms. *aa*, Minute aneurysms of branches of the cerebral arteries (natural size); *b*, Aneurysmal bulging of a capillary vessel, with fatty degeneration of its walls (enlarged).

Charcot and Bouchard considered that the formation of miliary aneurysms was a constant preliminary of cerebral hemorrhage; the aneurysms were found in seventy-five consecutive cases in which such hemorrhages were found after death. Charlewood Turner, however, considers it doubtful whether in some cases the hemorrhage may not be from direct rupture of the vessel due to inflammatory softening of the walls without saccular dilatation. Recent inflammatory lesions were the signs he noted in the cases examined. (Transactions of the Pathological Society of London, 1882, p. 96.)

Miliary aneurysms are not infrequently met with in cases in which there is atheroma of the larger cerebral arteries, but there is no direct relation between the two conditions—there may be atheroma without miliary aneurysms and miliary aneurysms without atheroma. There is a distinct relation of consequence between chronic Bright's disease and miliary aneurysms within the brain. "It cannot be doubted," says Gowers, "that Bright's disease is a cause of miliary aneurysms in persons after middle life and sometimes at earlier ages."

The great majority of cases of aneurysm of the minute cerebral arteries leading to intracranial hemorrhage—at least four-fifths of such cases—occur after the age of forty. The disease is for the most part one of the degenerative periods of life.

Lancereaux has, however, described the case of a lad, aged fifteen, in whom miliary aneurysms were observed in association with tubercular meningitis. An old spot of cerebral hemorrhage was also found. It seems that periarteritis with aneurysmal dilatation of the arteries can be produced (though very rarely) by the agency of the tubercle bacilli.

It seems probable that the minute arterioles in some situations may present dilatations due not to an inflammatory cause but to local fatty degeneration of their walls.

In the great majority of cases, aneurysmal dilatations of the arteries supplying the central nervous system, and their rupture under the pressure of the blood current, cause the phenomena of *apoplexy*. It is by no means improbable, however, that similar dilatations and ruptures of minute vessels in certain areas may be determining causes of other diseases, the pathology of which is yet obscure. The observations of Hale White lend probability to the view that such changes in the minute arteries or capillaries near the floor of the fourth ventricle may be pathological factors of Graves' disease. Those of Charlewood Turner suggest that in the motor area of the brain they may be determinants of chorea. Some observations tend to show that arterial dilatation and rupture within the spinal cord may occur in influenza or in sequence thereto, and may explain some of the nervous lesions of this disease.

During their period of formation the *diagnosis* of miliary aneurysms within the brain is impossible. The probability of their occurrence must not be lost sight of in cases of chronic Bright's disease, especially if symptoms suggesting atheroma of the larger cerebral arteries concur.

The *treatment* of ruptured miliary aneurysms is of course that of cerebral hemorrhage, which will be considered in a later volume.

Obliterating Endarteritis.

This has already been described in the section on arteriosclerosis. There is, however, a very rare variety of the disease which requires some mention. The internal coat of the smaller arteries of the upper and lower extremities is first affected, and afterwards the disease involves the larger arteries. These latter are felt to be hardened; the pulse in them is observed to be narrowed and diminished, and subsequently no pulsation can be detected.

The disease is a very rare one. The symptoms are coldness of the region affected with aching pains. In later stages these pains may be intense—the part may become hard and cold and in the end gangrenous.

There are no known causes for the affection which occurs mostly in middle life. A case has been recorded by Dr. Bertram W. Bond, of Englefield Green, of a boy, aged fourteen, in whom no pulse could be felt anywhere in the left upper extremity till the subclavian was reached—the radial and brachial arteries were mere cords. There were no notable symptoms except a sensation of "pins and needles" in the left arm and fingers. The collateral circulation was good, and the arterial conditions in the right upper extremity were normal. The observer mentions a similar disease in two other patients, aged twenty-three and twenty-four years respectively (*Lancet*, January 19th, 1895, p. 150). A case has been recorded by Mr. Pearce Gould in a patient aged nineteen, and another by Dr. Hadden in a patient aged thirty-five.

Calcareous Infiltration of Arteries.

The deposition of calcium salts in the course of chronic aortitis and arteritis has already been noticed. This deposition follows the exudation, the fibrous proliferation, and the fatty degeneration in the layers of the internal coat and the adjoining middle coat of the vessel. In certain cases, however, the deposition of lime salts takes place primarily in the muscular fibre cells of the middle coat. These are seen first as opaque granules, which coalesce to form a patch and subsequently a cylinder of brittle material, which sometimes assumes a crystalline appearance, occupying the place of the middle coat of the vessel. This form of calcification is often marked in the femoral and brachial arteries and extends to the arteries of medium size but not to the smaller vessels nor to the smallest arterioles. Often in atheromatous subjects, the ordinary form of chronic

arteritis is seen in some situations, and this mere calcification of the middle coat in others.

It has been shown that such deposition of lime salts may occur as a process of metastasis. In a case recorded by Kuttner, in which incrustation of the smaller arteries occurred, the source of the lime salts was an acute caries of the vertebræ (the first dorsal to the last lumbar). Owing to chronic renal disease there was probably an impediment to the excretion of the lime salts. Except for the deposition of the calcareous matter, the walls of the vessel were found to be normal (Coats, "Manual of Pathology," p. 414). The calcified middle coat sometimes undergoes a true ossification.

Lardaceous Disease of the Arteries.

In lardaceous (amyloid, waxy, or albuminoid) disease, the deposition of the morbid material begins in the walls of the blood-vessels, especially in the coats of the smaller arteries, in the capillaries, and in the connective tissue. A frequent result is diminution of the calibre of the arteries so that the tissues supplied by these are rendered anæmic; hence atrophy and fatty degeneration of these tissues. Sometimes hemorrhages are the consequences of the change in the blood-vessels, especially in the intestines, where they are observed after death as isolated submucous ecchymoses.

DISEASES OF THE VEINS.*

Varicosity of the Veins.

Definition.—A *varix* is a dilatation of a vein or veins of a permanent character and accompanied by a thickened condition of the coats of these vessels.

There may be a simple dilatation (*phlebectasis*) of a vein which is caused by obstruction to the flow of blood; but this is not a true varix, that word implying permanence of dilatation. The term phlebectasis is also applied to a cluster of knotted and swollen varicose veins.

Etiology.

A varicose condition of a vein is caused by conditions that induce protracted or frequently repeated distention by obstacles to the flow of blood from the vein, such as compression from external constric-

* I desire to express my thanks for the efficient help afforded me in the preparation of this section by Dr. W. H. Kesteven, of Hillwood, Hendon, London.

tion or tumors, or by overdistention of the veins from there being forced into them more blood than they are competent to contain. There may be an hereditary disposition to varicosity of the veins, a congenital weakness of the vein-walls.

The veins of the surface of either the skin or mucous membrane are more prone to become varicose than those more deeply seated. The lower limbs are more frequently the seat of varicose veins than other parts of the body. This is, of course, owing to their dependent position and the consequent greater weight of the column of blood which the veins have to support. The vein in the lower limbs which is most frequently affected is the internal saphena. Varicose veins are also met with, in connection with hemorrhoids, in the rectum, where the vessels are but loosely supported by the surrounding parts and where, from their direct connection with the portal circulation, they are readily affected by any obstruction to it.

The spermatic veins are also frequently varicose, their swollen condition having been recognized under the name varicocele. The more deeply seated veins may also become varicose, but this is a much rarer occurrence than is the case with the more superficial veins. Pressure, as from tumors, etc., is the most frequent cause. The deep veins most commonly affected are the internal jugular, the vena azygos, and the prostatic veins.

Symptoms.

Varicose veins on the surface stand out with prominence more or less marked according to the amount of the varicosity. When they are close beneath the surface the skin seems to be thinned over them, and the blue mottled masses clearly show through it. To the touch they seem elastic, but the walls feel stiffer and thicker than normal. The incompetence of their valves is shown by the fact that the impulse of a cough can be detected in the leg, the wave passing readily through the dilated lumen of the vein. The enlargement of the vein is seen to be not only in the direction of lateral dilatation, or enlargement of the lumen, but also in the longitudinal direction—the vein seeming to be stretched and extended in length; the result of such extension being that it becomes convoluted or folded on itself, or twisted spirally, so that it may form a considerable mass. It is such a mass which has received the name of a phlebectasia.

Morbid Anatomy.

The Internal Structure of the Veins.—A description of the changes which take place in the walls of veins thus affected necessitates a

preliminary understanding of the normal condition of those walls in order that the changes may be localized and the departures from the normal standard specified. For this, however, there is not required any very minute description of the normal anatomy; for our purpose the following general picture is sufficient: The walls of the vein are divided, broadly speaking, into three layers or coats, namely, an outer, which forms the larger portion of the vessel-wall and is called the *tunica adventitia;* a middle coat, or *tunica media;* and an internal, or *tunica intima.* These layers are generally spoken of by their adjectival designations, "adventitia," "media," and "intima." The adventitia, when duly prepared and stained, presents under the microscope an abundance of coarse-fibred connective tissue with numerous cells. This connective tissue is arranged in interlacing septa, between which lies an irregularly arranged network of elastic fibres, some broad, some narrow, being more numerous on the inner than on the outer side of the adventitia. Among these elastic fibres are the larger vasa vasorum and the nerve fibres. There are also in this layer some bands of smooth muscular fibre arranged parallel to the axis of the vessel; these muscular fibres become more numerous as we approach the cardiac terminus of the venous system. Internal to the adventitia and in section appearing in the majority of veins to be about one-half the thickness of the adventitia is the media. This chiefly consists of bundles of smooth muscular fibres, anastomosing freely and thus forming a network. They are arranged in a circular direction or at right angles to the axis of the vessels. In the meshes of the network formed by the interlacing and anastomosing of the muscular fibres, is connective tissue, which encloses elastic fibres coursing parallel to the axis of the vessel and sometimes placed obliquely to that axis. Here also are found the smaller divisions or branches of the vasa vasorum.

Within the media is placed the intima, which consists of a fenestrated membrane, or rather a layer having a fenestrated appearance in microscopical sections. This appearance of fenestration is due to the sectional interruption of the continuity of the elastic fibres, which being much and complexly folded suffer irregular mutilation in the process of cutting. These elastic fibres run parallel to the axis of the vessel, and the fenestrated appearance shows, therefore, more plainly when the section is transverse to the vessel. With this elastic layer there is a hyaline substance, containing nuclei more or less patchy and irregularly distributed, which forms the connection between the intima and media. Internal to the fenestrated or elastic layer are seen cells and nuclei, both muscular and connective, varying in amount in different veins, and on their innermost surface is the endo-

thelium or lining epithelium of the vessel. In different veins these layers vary not only in relative thickness but in general size and amount. In some the intima is thicker, in some thinner; in some the elastic elements predominate over the muscular in the media, and *vice versa;* in others the adventitia is much reduced in thickness; but the three coats or layers are to be found in all veins likely to come within the purview of our subject. Another important element in the structure of the vein from our point of view is the valvular apparatus. We cannot do better than transcribe Epstein's account of these structures:

"The valves of the veins belong, as is well known, to the class of pouch valves. The valve flaps consist of stout fibrillar connective tissue and elastic fibre bundles. Both surfaces of a valve flap do not exhibit an exact resemblance; for while in the surface presented to the periphery, or rather met with in passing from the direction of the capillaries, there is beneath the endothelium a fine network of elastic tissue; under the endothelium of the centrally presenting surface or side nearest the heart there is a connective-tissue layer in which only faint traces of elastic fibres are recognizable. . . . The valve flaps are always arranged in pairs, so that as a rule both pouches are placed opposite each other, but one pouch is generally somewhat displaced upward or downward. The valve flap forms a V-shaped sac or pouch in connection with the vein wall. The V-shaped dilatations which are formed in the vein wall by the attachment thereto of the valve flaps extend in a cardiac or central direction rather beyond the free edges of the flaps. They consist largely of muscular tissue, the fibres of which are arranged nearly parallel to the direction of the dilatation or circularly or perpendicularly to the axis of the vessel, which fact can be easily made out in a successive series of sections. The smooth bundles of muscle fibres lie between elastic networks, which are formed by extensions of the elastic membrane of the intima. The dilatations contain an abundant quantity of connective tissue and cells. To a small extent these muscular fibres of the dilatation of the vein wall pass into the neighboring part of the valve flaps. This sinuously bulged section of the vein wall placed opposite the valve flap shows a further peculiar structure. Here we find under the endothelium a layer of longitudinally arranged bundles of muscular fibres which are penetrated by fine branchings of the internal elastic layers and appear to be embedded in a homogeneous connective tissue. In this are some cells which I feel impelled to consider as connective-tissue substance. Besides and under these in the media a scanty number of bundles of circular and of longitudinal muscular fibres are encountered.

"These muscular fibres, which are set parallel to the axis of the vessel, travel upward or toward the heart, a little beyond the level of the free edge of the valve pouch and then gradually disappear. The circular fibres here appear in the media, and as the dilatation diminishes become stronger and more marked, while at the same time the intima again assumes its usual appearance. On the peripheral side the longitudinal muscular fibres of the media extend to the valvular dilatation into which they penetrate. To the same extent on the under surface of the valvular dilatation the muscular bundles peculiar to the media are carried; also the greater part of the longitudinal fibres of the intima vanish on the valvular pouching, but some reach beyond it. They are placed here in a layer of connective tissue which inclines toward the dilatation in a peripheral direction and vanishes within a few millimetres thereof. In this are found a few elastic elements, which are manifestly part of the internal elastic layer and further downward unite therewith. The valvular dilatations are rich in capillary blood-vessels (vasa vasorum). We must add to this that the outer wall of the dilatation in comparison with the vein wall, both above and below, appears to be thinned, and that all being clear, the blood pressing downward on the upper surface of the valve is shut out by their meeting endothelial surfaces."

Varieties.

As leading to a more exact acquaintance with the morbid changes which are found in phlebectasy (varicosity of the veins), we may adopt a classification of the disease based upon the different appearances presented by these abnormal conditions. These variations in form have been named by Epstein: (1) The cylindrical enlargement of the larger veins; (2) the cirsoid or serpentine enlargement; (3) the varicose phlebectasis.

1. *Cylindrical Enlargement.*—This variety exhibits an increase in the size of the lumen of the vessel but no contortion of form; the vein is simply enlarged and the increase in size is only moderate in amount, and but slight changes are found in the vessel walls. These are principally marked by a slight infiltration of the adventitia and media with small cells, while the intima remains unchanged. It is only as the cylindrical condition passes into the more varicose condition that the changes in the intima make their appearance; these consist in the formation of a layer of connective tissue of uniform thickness which has its seat immediately under the epithelium.

2. *Cirsoid or Serpentine Enlargement.*—In this variety the changes are much more marked. The intima exhibits throughout the affected

portion a sub-endothelial layer of connective tissue of varying thickness in correspondence with the folded, bulged, or knuckled condition of the vein. We are driven to the conclusion that this thickening is a product of a compensating condition of phlebitis, which causes the dilated lumen to be again narrowed to the normal size. This conclusion is further confirmed when closer observation brings out the fact that the media to a greater or less degree exhibits spots of thinning or of bulging outward, while the intima at the same time is thickened in proportion thereto, so that the normal form of the lumen of the vessel really suffers no interruption. The process to a certain extent resembles what is seen in arteriosclerosis, and the resemblance goes further still, for calcareous deposits begin to be formed in the intima and perhaps even in the media; later on we observe a hyaline degeneration of the newly formed connective tissue of the intima, and when this takes place the elastic muscular layer of that coat is affected in the same way. In the media the normal muscular layer is in many places interrupted, these interruptions corresponding to the bending and knuckling of the veins which are much contorted. In this way the media is often thickened also, but this change is due to the contorted state of the venous tubes, the many twistings and turnings of which the vein walls must necessarily follow. But besides the changes thus caused, the media shows other considerable variations in its thickness. At one point the circular muscular layer is thinned and consequently the connective networks which penetrate it are thickened. At another the circular muscle layer is widened and the muscular elements compressed so that only single elastic fibres are perceptible among them. In consequence of the strain on the vein wall, the latter becomes more like that of an artery, without, however, having the same thickness as an artery of the same calibre.

There is indubitable hypertrophy in the sense that notwithstanding the widening of the lumen, the vein wall is also thickened. There is an hypertrophy of the media, which, in points where the widening is excessive, gives place to an atrophic condition. These changes in the media are accompanied by an increase in the number of the vasa vasorum in the adventitia as well as an infiltration of small-cell structure in all the layers of the vein wall.

3. *Varicose Phlebectasy or Varix.*—This third variety takes the form of spherical dilatations, forming nut-shaped swellings which stand out among the convoluted twistings of the former variety. The formation of these varices is due to the position of the valves; the vein wall on one or the other side of the vessel yields in the position of the dilatation above described. In them the various coats

present the following appearances: There is a considerable increase in the connective tissue of the intima to be found all over the dilated portion, but along with this connective-tissue thickening of the intima we find a diminution in the muscular elements of the media, with a distortion in the direction of the normal muscular fibres and, in the most dilated portion of the varix, the media seems entirely to disappear so that it is only by the employment of reagents that a few remnants of its elastic fibres can be detected. The adventitia is penetrated with a small-cell infiltration; it contains all the vasa vasorum of the vessel wall, and for these reasons does not appear strikingly thinner than normal. From the appearances thus detected it becomes clear that varicose enlargement of the veins is due to an atrophy of the media and hypertrophy of the intima and adventitia, that this hypertrophy is due to a compensatory endo- and periphlebitis, and that these changes further produce a lengthening of the vessel. The result of varix, as far as the valves are concerned, is to destroy their utility and to allow the blood stream to be thrown back both into the lateral branches and into the main stem, thus increasing the pressure in parts which the valves would normally protect.

Effects.

It is commonly held that the dilatation of veins impedes the return of blood from a part. This leads to chronic congestion and œdema with ulceration, eczema, and induration of the tissues as results. That these conditions are frequently met with in connection with varicose veins is a fact, but that they are absolutely related as cause and effect is highly improbable. These conditions are met with independently of a varicose state of the veins, and such state may be present without them. The dilatation of a vein is sometimes attended with a thickening and pigmentation of the skin over it; more often the skin is thinned by the constant pressure. The alteration of the vein walls, the slowing of the circulation within them, and their prominence, which renders them liable to contusion, often lead to the formation of thrombi within them.

Treatment.

This may be either palliative or curative.

Palliative treatment consists in obviating the increased intravenous pressure caused by position, strain, muscular action, etc.; in affording external support to the vessels by well-fitting elastic stockings or a carefully applied bandage, or in improving the patient's general con-

dition by astringent tonics or by cardiac tonics where these are indicated.

Curative treatment consists in the obliteration or removal of the diseased vessels. It is indicated only in a minority of the cases; the multiplicity of varices and the simultaneous affection of the deep veins frequently render operations performed upon superficial veins disappointing in their results. Where, however, a single, limited varix exists, or one particular varix is a source of constant pain unrelieved by palliative treatment and disabling the patient, or where an enlarged vein leads directly up from a chronic ulcer which resists other therapeutic measures, operative treatment of varix is called for. Many modes of radical cure have been proposed, but only three require notice.

Acupressure.—By passing a hare-lip pin beneath the vein, placing a piece of gum bougie over the vessel, and then throwing a silk thread in a figure-of-8 over the ends of the pin, the walls of the vein can be held in apposition and a certain amount of traumatic phlebitis be excited which may lead to the occlusion of the vessel.

One or many pins may be employed as may be required; they should be withdrawn in about a week unless marked irritation is excited earlier, and they may be left longer if the local irritation be very trivial; care should be taken not to pierce the vein, as that would be liable to excite more acute phlebitis. The operation often fails to obliterate the vein. Two pins may be introduced close together and the vein may then be divided subcutaneously; this renders the result more certain.

Ligature.—Through an incision, about half an inch long, down to the vein, a ligature may be passed around the vessel and tied. Silk soaked in carbolic acid solution (five per cent.) is the best material for the ligature; but catgut may also be used.

Excision.—By making an incision along a varix the vein may be exposed and dissected out. A double ligature should be tied around each branch of the vessel before it is divided.

All these plans of treatment should be performed with antiseptic precautions lest spreading phlebitis be excited. Intravenous injections of perchloride of iron or carbolic acid, or the application of the actual cautery or hot iron, are not to be recommended. Where a single pouch of a varicose vein demands treatment, excision is the best operation; where it is desired to cure a number of varices or one varix of considerable length, the multiple antiseptic ligature is to be employed, or, if the surgeon prefers it, acupressure.

Phlebitis.

Morbid Anatomy.

Inflammation of the veins in any serious degree does not occur from the ordinary forms of irritation. This is a matter of common experience—for contusions of the surface, necessarily attended by violence done to the superficial veins, and lacerated wounds often involving the venous channels, deep as well as superficial, heal usually without any symptoms to show that any morbid conditions of the veins have been produced. Exceptionally, however, the contusion of a vein has produced phlebitis and subsequently obliteration of the vessel. On this point precise experimental investigations have been made by Vaquez ("Phlébite des Membres." Clinique Médicale de la Charité. Paris, 1894). This observer introduced a sterilized needle into one of the exposed large veins of a dog, and, after having interrupted the blood flow, scratched with its point the lining membrane of the vessel. At the end of two hours no trace of clot appeared to the unaided vision on the internal surface of the excised segment on which the scratchings had been practised. Microscopical examination, however, showed that some epithelium had become denuded, and where the laceration had extended to the middle coat there was a fibrinous deposit containing both white and red blood corpuscles. At the end of twenty-four hours in another experiment there was abnormal vascularization of the external coat with dilatation of the vasa vasorum, the minute coagula on the inner coat presenting much the same characters as before. On the fourth day the vein appeared perfectly normal, and every trace of coagulum had disappeared.

After some other forms of irritation (as for example cauterization by a heated needle) no coagulum whatever was produced. In cases where a vein had been tied by a catgut ligature which was removed after the lapse of five or ten minutes, though a temporary parietal coagulum was observed, every trace of lesion had disappeared on the third day from the operation, and the vein was completely restored to the normal. All these experiments were made under strict antiseptic precautions.

In the inflammation of a vein which is of distinctive form and betrayed by symptoms during life the post-mortem evidences are these: the vein may be seen to be greatly dilated; the internal coat shows in some portions a loss of its smooth lining membrane, the epithelium having desquamated; its layer of fibrous tissue is infiltrated, and under the miscroscope are seen abnormally widened meshes. The external coat may be found greatly thickened and infiltrated with cells,

and the vasa vasorum may present dilatations and engorgements. The middle coat is sometimes unaffected; it is obvious that the lesions of phlebitis affect more the external and internal coats than the middle coat.

The most important post-mortem feature in the majority of cases of phlebitis is the clot which is observed within the diseased and dilated vein.

Thrombi and their Mode of Formation.—The coagula found after death within the vein may be of varied character. There may be a *red thrombus*, the corpuscles, both red and white, being entangled in the meshes of the fibrin: such occurs when clotting takes place in blood at rest. Or there may be a *white thrombus*, as occurs when blood is in rapid motion, the red corpuscles being carried away in the current. When such a coagulum extends, some red corpuscles may be caught in it and staining occurs. Sometimes the red and white occur inalternate layers—the thrombi are then stratified or mottled.

The shape of a thrombus varies according to the situation in which it is formed, and according to the influences which produce it. A thrombus formed in a venous sinus is simply a flat coagulum. Thrombi which are formed within veins under conditions of exhaustion are frequently developed in the first instance in a portion of the vein near a valve (Lancereaux), and the moulds of the venous valves may be observed upon their surface, the other extremities being conical pointed or presenting the form of the head of a serpent (Charcot and Ball). In some cases the coagulum forms at the spur of a bifurcation of the vein; then the clot extends into and blocks the smaller branch and occurs in the form of a cylinder or cone in the larger.

Although it is usual for a clot to commence at a point of the vein where a branch joins it or else at the situation of a valve within a vein, this rule is not absolute.

The following are the changes which may take place in a coagulum (thrombus) formed within a vein:

(1) *Softening.*—The centre of the clot or the conical extremity of it may become changed into a grayish pulp, and then gradually the whole may become softened or liquefied. The softened material is carried into the general circulation, but it may be quite non-irritating and may give rise to no symptoms. If the phlebitis and the associated coagulation are due to infective agencies, these may give rise to suppuration in the clot, and the pus and micro-organisms, being transported to distant organs, may cause abscesses in the tissues to which they are carried; or collections of micro-organisms may be thus carried in the absence of pus, and the conditions of disease will vary according to the infective agency thus transported.

By mechanical means or during the process of softening, portions more or less large of the clot may be detached and carried in the current of the circulation to the right chamber of the heart, and thence, urged by the systole of the right ventricle, may block the trunk or the branches of the pulmonary artery, giving rise to pulmonary embolism. At the post-mortem examination the appearance of the clot may indicate the situation whence it is derived. The coagulum found in the pulmonary artery may show the imprint of the valves of the vein from which it has been detached; and the fragments observed free in the pulmonary artery and its branches may so correspond with other portions found in the diseased vein itself as to afford convincing evidence that they have become detached from the latter.

(2) *Calcification* in the clot may occur. Thus *phleboliths* are formed; these consist chiefly of calcium phosphate with, in small proportion, calcium and potassium sulphates, and about twenty per cent. of proteid matter. They may mark the site of an occluded vessel or may lie free in the channel of a vein attached to its wall by a fibrous pedicle. In some cases the wall of a vein becomes indurated in spots which appear whitish, and in some of these situations the endothelium may have disappeared. These spots may undergo calcareous incrustation. In fact there may be changes analogous to those of atheroma of the arteries, though the yellow fatty sub-endothelial change is not observed. This calcareous venous atheroma affects mostly the iliac and femoral veins.

(3) *Organization* of the thrombus may occur in whole or in part. The diseased vein may be completely blocked, the affected portion being converted into fibrous tissue, and the vessels in proximity becoming dilated; or the thrombus may contract toward one side of the vein, causing a local fibrous thickening.

Vaquez has found that soon after the formation of a thrombus capillaries begin to appear at the spot of endophlebitis. These capillaries are in communication with the vessels of the external coat; they are surrounded by cellular elements which form around them an embracing cylinder. The cellular elements become enlarged and proliferate and new cells multiply in the internal coat, the thickness of which gradually increases. The capillaries which have arrived at the internal coat progressively advance into the interior of the coagulum. They seem to take their origin from the lacunæ or sinuses described by Cornil and Ranvier as occurring in the deeper layers of the internal coat. The gradual disappearance of the softer elements of the clot is due to the granular and fatty degeneration of these induced by the phagocytic action of the leucocytes; this softened material is carried away by the newly formed vessels.

The external coat of the vein presents changes at the earliest periods of the formation of clot within the vein; as the clot forms the coat becomes thickened, its connective tissue proliferates, and its vasa vasorum present evidences of inflammatory changes. The middle coat, which resists for a longer time than do the external and internal coats, becomes at a later period hypertrophied. Still later it becomes atrophied, the external and internal coats being merged together. The shrunken vein is at last represented only by a fibrous cord.

Micro-organisms in the Lesions of Phlebitis.—Doléris described (in 1880) micrococci as present in the internal coat of some of the large veins, such as the femoral, in certain cases of phlebitis occurring in the course of puerperal fever. Widal found in the diseased portions of the walls of the veins, in a large number of cases of puerperal phlebitis, micro-organisms of the same character as those found within the uterus, namely, chains of *streptococcus pyogenes*. Other observers have found the bacillus tuberculosis in tubercular phlebitis. The pneumococcus and the bacillus coli communis have also been observed, and also the bacillus of Escherich in an experimental investigation. Hutinel found micro-organisms within the veins in cases of phlebitis occurring in the course of typhoid fever. The presence of infective agencies in the external coat of the diseased vein has been demonstrated by Widal and others. It has been noted that these affect especially the vasa vasorum (Widal, Boinet, Battone, Vaquez, and Thérèse).

Modes of Origin.

At a very early period, when the clinical and pathological phenomena of phlebitis became subjected to close study, there were differences of opinion as to the relation which the clot formed within the vein bore to the disease process. Cruveilhier considered that the coagulation of the blood within the vein was a consequence of a morbid condition of the internal coat of the vessel: "Le sang, chargé de principes irritants, irrite les parois veineuses, et le premier phénomène de cette inflammation c'est la coagulation du sang." Virchow, on the other hand, concluded that the formation of the clot preceded the inflammation of the wall of the vein, and this, acting as a foreign body, was the cause of such inflammation. These views met with much acceptance. Mr. Pearce Gould says: "It is not known that a thrombus may form in a vein independently of inflammation of its walls and subsequently excite phlebitis" (Treves' "Manual of Surgery," Vol. I., p. 369. Cassell & Company, 1886).

This explanation of Virchow, that the clot is first a passive formation and only secondarily an excitant of inflammatory action in

the walls of the vein, seemed very plausible when that large group of cases of phlebitis occurring in association with enfeeblement of the circulation was considered. In this group are the phlebitis of anæmia and chlorosis, and that of the subjects of wasting disease such as tubercle and cancer. The opinion held was that the formation of the clot is entirely, or almost entirely, a mechanical process; the flow of blood through the vein becomes so retarded that coagulation takes place and the thrombus is gradually formed. Lancereaux indorsed this theory. He said that the thrombi occurring in the wasting diseases (*thromboses marantiques*) are always formed at the situations where the blood has the maximum tendency to stasis, *i.e.*, in those portions of the veins where the walls cease to adhere to the fibrous tissue in their vicinity; where, therefore, the aspirating influence of thoracic expansion tends to diminish or disappear and where the force of cardiac inflation becomes annulled.

Subsequent investigation, however, has shown that there may be complete arrest of the blood current within a vein for long periods without the occurrence of coagulation. The blood in the jugular vein of a horse may be enclosed between two ligatures and thus rendered motionless, and yet for at least an hour and a half it will not coagulate *in situ* though the blood withdrawn from the enclosed portion clots rapidly (Thackral, Lister, Sandamore). "Within the body, if a vessel be ligatured carefully in two places, the middle portion remaining in connection with the living tissues, then the blood may be kept fluid from twelve to fifteen days" (Coats). Coagulation in the vein does not occur until some morbid changes have taken place in the internal coat of the vessel (Brücke, Zahn, Baumgarten, Vaquez).

It is now generally agreed that the immediate cause of the coagulation of the blood is the reaction of a ferment upon the soluble fibrin elements. Buchanan in 1845 concluded from his investigations that fibrin was not a substance which coagulated spontaneously, but that its precipitation in the solid form was the effect of certain agents such as casein or albumin. The coagulating ferment may be derived from the white blood corpuscles, or from some modification of them. So long as the white corpuscles are in normally vital conditions in the blood, the latter remains fluid. If the white blood corpuscles are in contact with dead matter or become in any way themselves devitalized, a fibrinous coagulation forms. Some observers have concluded that the coagulation is effected by a special form of white corpuscle (blood platelets or disklets) which exists in the blood (Hayem, Bizzozero, Eberth, and Schimmelbusch). The coagulating ferment, however, may be derived from other sources than the blood corpuscles. Bauschenbach obtained a soluble ferment which deter-

mined coagulation from the cells of the lymphatic glands, and Foà and Pellicani from fresh brain substance. It seems most probable that the ferment which determines coagulation and the formation of thrombus within a vein may be derived from the corpuscles in the blood current or from material conveyed into the wall of the vessel from without.

The important agency of the wall of the vessel is shown by many facts. Although, as I have said, contusion of a vein rarely causes phlebitis, exceptionally it does so. Verneuil noted periphlebitis and thrombosis as occurring in the femoral vein after prolonged manual compression of the vessel in the thigh. Langenbeck and others have recorded cases of phlebitis with thrombosis occurring from denudation of the veins of the neck. Reference has already been made to the experiments of Vaquez on the living animal, which showed that, though some parietal coagula were observed after the temporary ligature of a vein, there was a rapid return to the normal, and the lesions were practically healed on the third day. The same observer found that, if, before the application of the ligature, the vein was exposed for a considerable part of its course, and isolated from surrounding tissues by a thin layer of sterilized cotton-wool, the parietal coagula were more extensive and more persistent, and the restoration of the vein to the normal was a slower process.

Lancereaux has described the case of a young woman who suffered from suppuration of the left ovary and the left hip joint after parturition, and in whom the left utero-ovarian vein showed a collection of pus between the internal and middle coats. Corresponding to this area there was seen in the interior of the vessel a coagulum consisting partly of pus and partly of a fibrous thrombus. No doubt the phlebitis and the thrombosis were caused by the infiltration of the pus-producing micro-organisms or of their soluble toxins. Weigert has demonstrated the passage of infecting agencies from the external to the internal portions of the vessels, and many observers have shown the presence of infecting micro-organisms in the interior of the vasa vasorum. Vaquez has recently formulated the definite conclusion, that there is no persistent clot unless there is a persistent lesion of the vein.

I consider it in the highest degree probable that the phlebitis of the specific infective diseases is due to the transport of micro-organisms to the minute arterioles supplying the wall of the vein, the formation of thrombi being due chiefly to lesions of the internal coat and of the endothelium effected by the toxins of these micro-organisms.

Analogy with Chronic Arteritis.—Borel, in 1859, showed that

atheroma of the veins was sometimes to be found in conjunction with arterial atheroma, and his observations were confirmed by many others. In exceptional cases lesions of the vein are found, completely analogous with those in an atheromatous artery, and the causes are alike for both. There is good reason to believe that a lesion of the nutrient arteries of the vessel (the vasa vasorum), whether of the artery or of the vein, is the initial cause. In the case of the artery the force of the blood current and the greater movements of the muscular wall of the vessel prevent coagulation unless there is such softening as an aneurysmal pouch forms.

In the vein where the flow is slow and equable, clotting is more readily induced when the coagulating ferment permeates through the internal coat. The energy of the blood current and the anatomical differences in the walls of the two sets of vessels are the causes which make the pathological picture different in the two cases. We have seen that the disease-changes in the pulmonary artery are identical with those in the aorta. Atheroma is rarely seen in the pulmonary artery except in cases of over-tension within the vessel, as in obstructive lung diseases and in diseases of the mitral valve, especially mitral stenosis. Well-marked atheroma is not infrequently seen in the pulmonary veins; Barr, of Liverpool, has observed pronounced atheroma in them, in case of mitral stenosis. It is to be remembered that the pulmonary veins are very muscular organs and their forcible contraction precedes the auricular systole. In arteritis, whether acute or chronic, we find patches of inflammatory disease and degeneration in areas which probably correspond to tracts fed by the vasa vasorum. In disease of the vein, in most cases, thrombosis is an early result, and the changes in this thrombus, whether resulting in its softening and removal or in its vascularization, fibrosis, and atrophy, present a series of events different in appearance though initiated by like causes.

Pathological Groups of Phlebitis.

These are: 1. Non-obliterating phlebitis; 2. Obliterating phlebitis; 3. Recurring phlebitis; 4. Chronic phlebitis.

1. Non-obliterating Phlebitis.

The word "obliterating," as here used, refers to the lumen of the vessel, and therefore, as regards the blood stream, means "obstructing." It does not necessarily imply destruction or "wiping out," so to speak, of the vein itself. There are two kinds of non-obliterating phlebitis.

(*a.*) The septic or infectious form of phlebitis is due to the materies morbi which induces the general disease. We have all the symptoms of purulent infection, and numerous veins are affected. Traumatic or surgical phlebitis arises from external infection; in other forms of phlebitis the infection is derived from within. Varicose ulcers may give rise to this form of phlebitis. It also arises in the puerperal condition, sometimes as late as two months after the confinement. This multiple superficial phlebitis arises in some cases of phthisis and is not infrequent in gout.

(*b.*) A non-obliterating phlebitis occurs in the stage that precedes the full obliteration of the lumen of the vein; when recognized, this form is called pre-obliterating phlebitis, but it is most commonly overlooked. This form really constitutes the preliminary stage of phlegmasia alba dolens.

Symptoms.—In the cases of non-obliterating phlebitis the symptoms always seem out of proportion to the general condition or accident which has given rise to the mischief. The temperature is high; there are rigors, profuse sweats, pallor of the face, and marked dryness of the tongue. The limb in which the veins are inflamed becomes painful; sometimes the pain is diffused, at others it follows the course of the vein or veins affected. With this there appears an œdematous swelling of the part, at first rose-colored and accompanied by a certain amount of lymphangitis.

Course.—However the poison may have reached the vein, the result is an inflammation of the coats of the vessel. It may be that this inflammation is limited to the vessel, itself, or the contiguous perivascular cellular or connective tissue may be involved. The inflammation may spread further, infecting the lymph spaces and afterwards the lymphatic trunks and even the glands. In this way limbs may become the seats of phlegmonous inflammation, of abscess, or of purulent lymphangitis. In mild and favorable cases the mischief is limited to the vein or to the limb, and, after a short time it disappears. In cases where the poison is much attenuated the vein or veins affected are simply painful for a few days; the skin over them appears reddish in color with a tendency to a bluish shading on either side. In all these cases there is in fact more or less periphlebitis, and it is the thickening caused by this around the vein which has given rise to the idea that the vein is obliterated or obstructed. Veins thus affected are all more or less liable to become obliterated, but they do not necessarily do so, and recovery may take place without their having lost their permeability. The cases which do pass on to the stage of obliteration, those, namely, of pre-obliterating phlebitis, are generally the ones which occur in patients in the final stages of pulmonary

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tuberculosis or toward the end of cancer; and these cachectic cases usually afford the best types of pre-obliterating inflammation of the veins.

2. *Obliterating Phlebitis.*

This pathological group must also be subdivided into two classes, viz., obliterating phlebitis of the smaller veins, obliterating phlebitis of the larger veins. The reason for this subdivision is that when the phlebitis results from an acute septic action, its evolution is rapid, and only the small veins can be so affected as to contain coagula, leading to complete obliteration. When, however, the dose of the poison is smaller or its character less virulent, and its action is in consequence less acute, in order that obliteration of the larger veins may occur there must be a certain amount of deterioration in the veins themselves. This is the case in phlegmasia alba dolens.

The obliterating phlebitis of the smaller veins is met with in two distinct clinical varieties: one, in which the phlebitis is a local complaint and results from traumatism or spontaneous rupture of a vein, such, for example, as that met with as a consequence of phlebotomy, or in cases of rupture of aseptic varicose veins in pregnant women; the other, in which the disease is the result of processes which are more distinctly septic in their nature.

Symptoms.—In the subject of varicose veins the disease first shows itself, after a sudden or excessive exertion, by a sudden, sharp pain like the stroke of a whip. It is localized at the point where the mischief occurs. The varix affected presents a dark color, and, after a few days, the appearance is that of a bruise, elliptical in shape with the long axis in the direction of the vein. It feels hard to the touch. This hardness does not disappear as the inflammation decreases, but becomes gradually more limited in extent, finally leaving a small hard knot which is due to the presence of a thrombus. These knots are sometimes very tender and remain so. The ecchymosis disappears in about two weeks. A varicose rupture is not a common occurrence in ordinary cases. As pointed out in treating of this complaint, the walls of veins thus affected are subject to a kind of compensatory phlebitis which rather strengthens them, but in cases of pregnancy there is not time for this to obviate the phlebectasy, and the veins are more easily ruptured. This condition is not limited to the lower limbs, but may affect the veins of the upper extremities and those of the mammæ.

The general symptoms of the more septic form of phlebitis depend more upon the particular nature of the veins which excites the mischief, and therefore must be treated of in the description of the clini-

cal aspects of the case rather than at this point, but we may notice here that the outbreaks of this form of phlebitis depend to some extent upon the exacerbation of the peculiar malady of which they are one form of development. The veins of the lower limbs are most frequently and primarily attacked, notably the internal saphena, but later on we see the veins of the upper limb, the median cephalic and median basilic, affected. Sometimes even the jugulars are implicated.

In every case the phlebitis announces itself by pain at the point attacked. We then find very shortly a hard venous cord and a more or less extended œdema, which is never, however, very extensive. There is also a temporary redness or blush about the affected vein which is but temporary.

Obliterating phlebitis of the larger veins, long known by the name phlegmasia alba dolens, will be considered below (p. 620).

3. Recurrent Phlebitis.

In this form there is the reappearance in a vein previously attacked, and seemingly cured, of an inflammatory process.

This does not include the simple recurrences of pain in the site of an old phlebitis, nor those cases in which a fresh poison re-excites an inflammatory condition—as, for example, where an attack of inflammation occurs in an individual suffering from tuberculosis who has been the subject, at some former time, of phlegmasia alba dolens, and in whom it might happen that the tissues in proximity to the obliterated vein were attacked by the poison and a condition of periphlebitis of the venous cord was thus established. This might give rise to symptoms resembling recurrent phlebitis, but as the vein has been practically destroyed it can not really be considered such. Nor can we include among the cases of recurrent phlebitis those in which, for example, in successive pregnancies there is varicose rupture as described under obliterating phlebitis of the smaller veins, for here, in fact, there is a distinct cause for the fresh outbreak. The term "recurrent phlebitis," therefore, is limited to two classes of cases, viz., those in which the original disease, having arisen in the course of an infectious malady, has reappeared during an analogous outbreak, or during one of a different character; and those in which the phlebitis appears as a recurring complication of a general disorder, or of a local venous affection manifesting periodical aggravations of the primary disease.

Recurrent phlebitis of the first class is now rarer than it was, thanks to the greater cleanliness and the attention directed to hygienic conditions which has resulted from the introduction of antiseptic

precautions. Some doubt the possibility of the occurrence of this form of phlebitis if proper antiseptic precautions are taken, but a patient who has had phlegmasia retains in his or her own person a tendency to have it again which must not be lost sight of. In such, notwithstanding the utmost precautions, the disease may recur.

Gonorrhœal phlebitis is not infrequently recurrent. Phlebitis also recurs in influenza, in the later stages of consumption, and in patients with cancerous cachexia. In these cases the veins attacked are not necessarily those previously affected, and this is explained by the fact that the original phlebitis has obliterated the veins it involved, and the phlebitis which we now see affects those in which collateral circulation has been established.

Cases belonging to the second class of recurrent phlebitis are more commonly met with in the subjects of local and persistent lesions of the veins, or those in whom there is developing at slow rate some general malady, whether infectious or not in its nature. Gouty patients, especially those with varicose veins, are instances of this class of recurrent phlebitis. Varicose veins are specially prone to be the seat of recurring inflammation.

4. Chronic Phlebitis.

This is a persistent painful or tender induration of the vein walls resulting in atheroma.

Causes.—These are said to be alterations and obliterations of the vasa vasorum and so interference with the nutrition of the walls of the veins, resulting in an inflammatory condition.

The first indications of mischief are pain and induration of the vein. This condition is recognized under the name of *venous sclerosis*, and, becoming more marked, it constitutes an atheroma or degeneration similar to that met with in the arteries.

Clinical Groups of Phlebitis.

Phlegmasia Alba Dolens.

This may be defined as a painful general swelling, white in color, of a limb, and due to the obstruction of the veins thereof—a plastic phlebitis with lymphatic obstruction.

The lower limbs are most generally affected, and of these the left more frequently than the right.

The varieties, as classed by Dr. C. Hubert Roberts, are: 1, cases due to pressure; 2, cases associated with general disease; 3, cases of true septic nature; 4, cases of thrombosis apart from sepsis; 5, cases of thrombosis and sepsis combined.

Symptoms.—There is at first an elevation of the general temperature. This may be, and frequently is, overlooked. It occurs sometimes eight, ten, or twelve days before the first manifestations of a more visible character. Phlegmasia alba dolens occcurs notably in childbed, and its advent should be looked for in women in this condition. In typhoid fever it also sometimes occurs during the very early stages of convalescence, and also in the later stages of convalescence; the elevation of temperature which precedes it is very much of the nature of that which is seen in hectic fever, and any such sudden elevation should excite suspicion of this morbid affection. It sometimes happens in cases of chlorosis, and then the onset of white leg is accompanied by a very sudden, transitory elevation of temperature. Other precursory symptoms are creeping sensations, formication, "pins and needles," cramps, shooting pains, etc.; but these are not always present.

Pain is often the first symptom that attracts attention. It may sometimes be very intense, sufficient to cause vocal expression in cries and groans. The pains are sometimes felt simultaneously on the inside of the thigh, at the fold of the groin, and in the calf. Sometimes the pain assumes a neuralgic type, not corresponding in its situation so much with the veins as with the sciatic nerve. At first the pain begins as a feeling of weight, or as of pins and needles, or darting nocturnal twinges. It is sometimes accompanied by rectal and by vesical tenesmus. The pain in other cases is moderate, and, indeed, may be absent, or only detectable on palpation; Vulpian is of opinion that palpation of the calf will reveal its presence. As a consequence of these painful feelings, the limb is kept motionless by the patient. If it be the lower limb which is affected it is held in a position of extension, if it be the upper limb it will be in a semi-flexed position.

The most marked symptom is the œdema of the limb. The amount thereof must not be taken as indicative of the importance or the size of the vein which is obstructed, for it may be considerable in a case of saphenous obstruction, and moderate in one of occlusion of the femoral vein. The œdema is soft in character, and the skin is white and shining, sometimes waxy in tint. When there is a tendency to set up collateral circulation blue veins are sometimes to be seen standing out in contrast with the prevailing white, particularly in the posterior parts of the limb. That the obstructed vein or veins can be felt as cords in the dropsical limb is beyond doubt, but it is not advisable to try to find them; such searchings have been known to be fatal, and the information gained is entirely insignificant, especially when compared with the risk encountered in making the exploration.

In the classification of Roberts, given above, the first variety can hardly be considered as genuine phlegmasia alba dolens. It might with more propriety be considered as a dropsy pure and simple, in which pressure obstructs the veins and possibly the lymphatics, causing effusion of serum in the parts from whence the obstructed vessels should remove the blood.

In the cases associated with general disease the condition may be due either to multiple thromboses—as, for example, among the uterine veins—or to a septic influence injuring the endothelium and so giving rise to coagulation in the veins of the limb or limbs affected. To the former cause are due especially the cases of puerperal white leg, and it is in the puerperium that the disease is most commonly met with. The clots which cause the mischief in these cases escape from the enlarged uterine veins into the internal iliac, and become fixed at some point beyond the junction of the femoral vein, plugging the vein and stopping the blood stream. Besides the puerperal state other conditions render the body liable to this disease. Among these are convalescence from fever, especially typhoid, dysentery, disease of the rectum, malignant disease of the uterus, interference with uterine fibroids, arrest of the menses, etc. Cases of true septic nature are such as arise from septic thrombosis originating in the veins of the affected limb, such thrombi being caused by septic micro-organisms. The poison elaborated by these (the toxins) on reaching the larger veins may operate destructively on the endothelium of these vessels, and so cause, in the manner described when treating of thrombosis, the formation of coagula in them. The thrombi may contain numerous microbes.

There may be, however, simple thrombosis, the coagulum containing no infective micro-organisms. Such coagula, simply fibrinous, may be detached from a vein in which there is simple phlebitis and be carried in the blood current to be fixed to the wall of a larger vein. In a given case there may be septic (infective) thrombi in some parts and simple (non-infective) thrombi in others. Such may be formed in the pelvic or uterine veins in inflammatory diseases affecting either the uterus or its neighborhood, the rectum or the bladder, as in metritis, perimetritis, cancer of the uterus or rectum, etc. The greatest danger is that of pulmonary embolism.

Symptoms Indicating Implication of the Nervous System.—These may be manifested (1) in the early stages of the disease, (2) in the later stages, or consecutive to the malady.

1. In the *early stages* there may be paralysis of motion in greater or less degree. Trousseau long ago insisted that the powerlessness of the affected limb was disproportionate to the other symptoms. He

noticed that in some cases there was no power on the part of the patient to execute any voluntary movement—to extend or flex the toes or to use the muscles of the thigh. Graves, Troisier, and Velippel have made similar observations, and all agree that the interpretation of the facts is difficult.

Verneuil has found that certain deformities may arise in the early stages of phlebitis, such as talipes equinus and equino-varus. These cases differ from such deformities as have a congenital origin in the fact that the toes are rigid, motionless, or semi-flexed in the form of a claw. In congenital cases, on the other hand, the toes show but little deviation from the normal positions and are generally freely moveable or else in a position of forced extension.

There are also many disorders of sensation. It is well known that pains in the affected limb occur in cases in which there is no œdema or in which vascular obstruction is but slight. Graves considered that phlebitis might be of a neuralgic form. In some cases the affected limb is benumbed—there is a condition of anæsthesia; in others the slightest touch to the skin causes pain—there is marked hyperæsthesia. Firm pressure is sometimes more tolerable than slight frictions of the surface. Sciatica has been not infrequently noted, and observers have located the sensory disorders in the area of distribution of the superficial nerves. There can be no doubt that there are many disorders of sensation in the subjects of inflammation of the larger veins which cannot be explained by the mere obstruction of the circulation.

Again there are often disturbances of nutrition. The œdema may be counted among these. It is evident that the dropsy observed is not commensurate with the degree of obstruction of the trunk of the vein. There is a disturbance of the lymphatic circulation. Purpura and effusions of blood beneath the cuticle have been observed in some cases, and in others bullæ like pemphigus and localized ulcerations of the skin.

The disorders of the nervous system which are manifested in the early stages of phlebitis, or before the obvious signs of inflammation of the wall of the vein are observed, may gradually disappear. The prognosis in regard to them is for the most part favorable, but in some instances they are prolonged so that they come into the category of the affections we are now about to consider.

2. Nervous symptoms in the *late stages of phlebitis* or consecutive thereto. Manifestations of pain or weakness in walking may occur several weeks or even several months after all the local signs of phlebitis have ceased. In some cases paresis has lasted for years. A frequent sequel of phlegmasia dolens is a persistent aching of the

affected limb, aggravated by cold and damp and also by undue movement. A disturbance of local nutrition has been shown in some cases by hypertrophy of the limb, constituting an elephantiasis. There may be local enlargements of the veins in bunches—like masses of leeches, as Vaquez has said. Some authors have also described varicose ulcers as consecutive to phlebitis.

It is probable that all these nervous phenomena associated with and consecutive to phlebitis of the large veins are really due to a peripheral neuritis. Klippel (1889) considered that the cause of the alteration of the nerve trunks was their being bathed in the morbid serum effused in sequence to the venous block. It seems more probable that there is a neuritis of the sciatic or crural nerves due to an implication in the disease of their own vessels—the vasa nervorum.

Prognosis.—As regards the leg the prognosis is usually favorable, a persistent wasting of the leg is rare; but there are many uncomfortable sequels to the disease, as already pointed out.

Diagnosis.—When the peculiar swelling of the limb has begun, the diagnosis is not difficult; but in the earlier stages the symptoms of pain may be misinterpreted. Even in the lying-in woman there may be a difficulty in differentiating the affection from hysteria. The probability of the onset of phlegmasia dolens in the case of a lying-in woman who complains of pain, especially if there be variable numbnesses referred to the thigh and groin, even though symptoms of hysteria exist, should be present to the mind of the physician. The importance of the differential diagnosis in the case of the phlegmasia occurring in anæmia will be dealt with hereafter.

Treatment.—The most important point as regards treatment is to arrange carefully the position of the affected or threatened limb. It should be comfortably supported upon a pillow so arranged that the foot is on a slightly higher level than the recumbent trunk. A better plan is to use an inclined plane or trough well lined with cotton wool, this to be supported by a pillow. Remember that any accidental jerk or shock to the limb may be a real danger. A clot may be detached from the inflamed vein and pulmonary embolism, very probably fatal, may result. Some consider that the greatest danger of such embolism occurs when the thigh is flexed upon the pelvis. The recumbent position, with the limb elevated and motionless, should be maintained for a long period. It is not safe to permit the ordinary position of lying upon the side before the thirty-fifth day of the disease nor the sitting position until the fortieth. Even then abrupt movements must be forbidden, though gentle walking may be allowed.

Sleeplessness should be met by some preparation of opium. As

this condition is mostly due to pain it is no use trying hypnotics, such as chloral or sulphonal, which do not annul pain. Except in cases of opium-intolerance both pain and sleeplessness should be met by the use of this drug or its derivatives. If the heart's action seems feeble, either from the use of opium or from the exhaustion due to the condition, stimulants should be given. Tonic medicines to increase the appetite may also be used.

To the leg and thigh, supported by the mechanical means previously indicated, fomentations of various sedative kinds may be applied; in such case the wool-lined trough must be protected by an ample piece of oiled silk or waterproof tissue carefully and gently inserted under the limb. Many prefer a light application of cotton wool to any moist application. Any sedative liniment may be applied with a gentle hand. Whatever variety of fomentation or wrapping be made use of, it should be done without forcible rubbing. Once or twice a day a sponge containing warm water may be gently squeezed over the limb, precautions being taken to prevent dampness either of the bed or of the clothing. The limb should, when not being fomented, be kept wrapped either in soft flannel or in cotton wool for its entire length. Movement should be permitted only when the cause of the mischief—the obstruction and inflammation of the veins—has quite subsided.

Duration.—After the disease has lasted nine or ten days it usually makes no further progress but recedes, the pain and swelling diminishing. But the rate of recession varies much. In favorable cases several weeks may elapse before the disease disappears, while in others restoration to a normal condition may require a considerably longer period.

The nervous sequelæ occurring in some cases have been already described.

Anæmic or Chlorotic Phlebitis.

It is more than doubtful whether there be in reality any such disease as a phlebitis due *simply* to anæmia. The anæmic condition is itself imperfectly understood at present, at least as regards its etiology. Careful examinations of many cases in which death has occurred have shown the existence of a lurking disease such as tuberculosis or cancer; or an attack of influenza may supervene on an anæmic condition and the phlebitis arise in consequence. There is, however, a form of phlebitis intimately associated with anæmia or chlorosis. In these cases the disease generally attacks the lower limbs, the left more frequently than the right.

Symptoms.—Phlebitis may be preceded by a condition of fever.

There is no doubt that the phlebitis is accompanied by a rise of temperature, especially when obliteration of the vein or veins affected takes place. The temperature oscillates for two or three days about 102.2° F. (39° C.) and attains its maximum about the fifth or sixth day. There is a moderate amount of œdema and severe local pain, and the presence of collateral circulation is shown by some enlarged superficial veins. If, however, it be a more deeply seated and larger vein that is attacked, the œdema is more diffused and the collateral circulation is more marked. By the fifth or sixth day the pain is generally much diminished and the temperature falls rapidly, the condition resulting being that of phlegmasia alba dolens, as described above.

A peculiarity of the phlebitis of chlorosis or anæmia is the tendency there is for the complaint to attack both limbs. The second limb is usually observed to be involved in bilateral phlebitis on the eighth or tenth day, there being at this time an access of feverish symptoms. In these cases there is of course the risk of complications common to all forms of phlebitis, and there is special danger of pulmonary embolism and even of death as the acute symptoms subside, the reason of this being that, the pain having disappeared and the swelling showing signs of diminution, there is great difficulty in keeping the patient quiet. If, however, this precaution is carefully heeded, the danger is diminished and recovery takes place sooner than in the other forms of phlegmasia.

Diagnosis.—In the anæmic form of phlebitis there may be great difficulty in the diagnosis in exceptional cases. I well remember being called to the case of a young woman who was highly emotional and hysterical and who complained of severe pain referred to the left knee joint. At first I inclined to the view that all the suffering was subjective. I made, however, a careful examination of the joint and soon found that the popliteal vein was firm, evidently containing clot, and its course painful. My manipulation was gentle, but I had no sooner left the house, having enjoined most complete rest, than I was called back in extreme haste. The patient was blue and almost pulseless, the breathing short and shallow, and the conditions critical. There could be no doubt that a portion of the coagulum had become detached from the vein and was now plugging the pulmonary artery or one of its larger branches. Aromatic spirit of ammonia was at hand and was administered, sufficiently diluted, in teaspoonful doses every quarter of an hour. At the end of three or four hours there were signs of amendment. Liquor ammoniæ fortior in five-minim doses diluted in half a wineglassful of water was then given every hour, and later every four hours. There was eventually a complete recovery.

This case illustrates the difficulties of diagnosis which may occasionally occur and the paramount necessity of great care in the manipulation necessary for making such diagnosis.

Treatment.—This consists in absolute rest until there is certainty that the inflammation has completely subsided; until all tenderness has departed; and until the circulation through the affected vessel is apparently re-established, or the thrombus has become so organized and fixed that there is no longer any risk of detachment. Warm wrappings with occasional fomentations of the limbs affected, care being taken to avoid any rubbing or violent movement, are also useful. The pain of the onset if severe must be met by opiates.

Embolism of the Pulmonary Artery.

In phlegmasia alba dolens, in chlorotic phlebitis, and in any form of phlebitis of a large vein, pulmonary embolism may occur. A portion, more or less large, of the clot may become detached, may be carried in the venous circulation to the right ventricle, and be thence forced by the systole of the latter into the pulmonary artery or its primary branches. The symptoms resulting from such accident as this are pain at the heart with sense of extreme distress and faintness; rapid and irregular but feeble contractions of the ventricles, as felt by the hand placed over the precordium; gasping and short, rapid breathing with intense respiratory distress; the issuing breath is sometimes felt to be cold, the face and surface are observed to be livid and bluish or else ashy pale, the lips and mucous membrane being of a pale slate color; the skin is bedewed with a cold sweat, and the limbs are sometimes convulsed. Though the hand may feel at the precordium the struggling, irregularly acting left ventricle, the fingers may be able to detect no pulse at the wrist or only occasional irregular pulsations of the radial artery. In such a case death may take place in a few minutes. Death is not due to mere asphyxia, but in some degree at any rate to syncope. The mental faculties are preserved almost to the last. It is only in exceptional cases that there are any distinctive signs to be found on physical examination. A soft systolic murmur has been audible in the course of the pulmonary artery in some cases; in others the heart sounds have been feebly heard and muffled. There may be a more gradual onset of symptoms. Such occurs when a small fragment of coagulum is impacted in a primary branch of the pulmonary artery and serves as a foreign body upon which further clotting takes place. Then there may be remissions and exacerbations of the symptoms during the space of several days or even some weeks.

In the *treatment* of this alarming and dangerous condition the free administration of ammonia is, in my opinion, the best plan to adopt. Sir B. W. Richardson long ago recommended that the strong liquor ammoniæ, sufficiently diluted, should be swallowed at frequent intervals. The ammonia not only acts as a rapid stimulant to the heart, greatly superior to any form of alcoholic stimulant, but also, promoting the fluidity of the blood, it tends to prevent thrombosis. When there are signs of recovery from the extremity of danger, the patient being semi-recumbent, the Sylvester plan of artificial respiration may be practised with caution. The arms may be so lifted as to induce thoracic expansion (inspiration) and then brought down to the sides of the chest, a slight compression of the thoracic walls being made at the same time (expiration). These movements should be made ten or fifteen times a minute for an hour or two with intervals of repose. At the same time gentle massage of the legs and thighs should be practised, and heat applied by flannels or hot bottles. It may be necessary also to administer nutritive enemata.

Gouty Phlebitis.

Definition.—Phlebitis associated with and dependent upon the gouty condition and its action upon the vasa vasorum of the veins.

Locality.—It occurs most frequently in the lower limbs, but not necessarily in that limb which has been or is affected by ordinary gout. The disease affects the superficial rather than the deeper veins. It may appear one day at one spot and the next day at some other part of the limb, or in that of the opposite side; there is a tendency to symmetry in the arrangement of the patches. This symmetry and the apparent metastasis and recurrences are peculiar to the gouty form of the disease, and point strongly to the fact that it is an affection of the walls of the veins and not an initial coagulation of the blood within them.

Symptoms.—There are severe aching pains in the course of the vessels; this being often the first symptom and for a time the only one. It is followed by tenderness to the touch and the ordinary phlebitic hardness and firmness. The integuments over the veins (when the affected veins are superficial) are slightly thickened and marked with a dusky reddish flush. There is generally œdema, but this is less marked in character when the veins are superficial than when they are more deeply seated. In the latter case the limb becomes large and clumsy, sometimes mottled here and there by the distention of the cutaneous veins. It is stiff and heavy. It feels œdematous throughout, but is firm and brawny, the skin being tight and

not pitting easily. In this, gouty phlebitis differs from other forms, and the peculiar tense character of the swelling may almost be considered as pathognomonic of the disease. It may be recognized when the œdema is limited in amount, as for example when the obstruction occurs in (say) the lower portion of the popliteal vein. Constitutionally the disease is accompanied with fever not very severe (temperature circa 100° F.), or by an ordinary attack of gout more or less acute in character.

Course.—The condition may pass off—the circulation may be re-established through the obstructed vessel or vessels; whether or not this will occur, depends in some degree upon the size of the veins affected. In other instances the obstruction becomes permanent and the limb always remains more or less swollen and heavy. Even if the veins become pervious they remain more or less susceptible to recurrence of the inflammation and subject to aching pain after exertion or weather changes. When the obstruction is permanent the superficial veins tend to become dilated or even varicose. In this, as in other forms of phlebitis, there is the danger of embolism and many fatal cases have been recorded.

Treatment.—As in other forms of phlebitis the essential part of the treatment consists in rest. Fomentations and warm flannels are useful if applied carefully so as not to risk detachment of the thrombi. Medicines or treatment of an active character, such as by leeches, etc., are of very little use for the phlebitis itself. The gout may, and probably will, need some medical treatment and the ordinary remedies such as colchicum, alkalies, and alkaline drinks should be used. Dietetic treatment is more beneficial. It should consist in the diminution of food and especially of stimulants. Water should be drunk freely.

Rheumatic Phlebitis.

There is not sufficient evidence to prove that a phlebitis of purely rheumatic origin has ever been recognized and it is highly improbable that any such morbid condition exists at all. In those cases in which a so-called rheumatic phlebitis has been said to exist, sufficient care has not been taken entirely to eliminate the possibility of the venous mischief being caused by other and more definitely known causes of inflammation.

Septic Phlebitis.

Definition.—Inflammation in the coats of a vein caused by the action of definite septic poisons. In these cases the phlebitis appears as a complication of the original disorder. Among the septic phle-

bitides are those which are included in the following categories: Phlebitis of primary septicæmia, of secondary septicæmia, and of the infectious maladies.

Primary Septicæmia.—Erysipelas of the face has been known to produce inflammation of a cerebral sinus and phlegmasia dolens. These are not always fatal. The outbreak of phlebitis in this disease makes its appearance in successive paroxysms coinciding with an elevation of the temperature lasting from twenty-four to twenty-eight hours, with intervals of seven or eight days. These attacks assume an almost cyclical character. Phlegmonous tonsillitis, by its purulent infection of the blood, sometimes produces purulent phlebitis. In these cases the large swelling of the tonsil has been noticed to disappear suddenly without apparent effusion of pus. Some days later, vague rheumatic pains are felt with general malaise, palpitations, and a tendency to syncope. The fever reappears and careful examination reveals myocardiac complications. These symptoms fade away and all seems going right when, perhaps days afterwards, there occurs an outbreak of phlebitis in the left lower limb. It may be as long as a month after the cure of the tonsillitis, and all other symptoms of infection may have passed off when this occurs. The phlebitis causes phlegmasia alba dolens which, however, disappears without further complications and the patient entirely recovers. Gonorrhœal poison is another cause of phlebitis by its occasional primary or septicæmic effects. The phlebitis in these cases nearly always coincides with gonorrhœal rheumatism. It attacks usually the large veins of the lower limbs, but it has been known also to affect the upper limbs.

Secondary Septicæmia.—The best-marked cases of this variety of septic phlebitis are to be met with in tuberculosis. The disease takes in these patients different forms. In the first form, which is rare and even exceptional, we see scattered coagulations in the superficial veins of the left lower limb but no production of phlegmasia. In other cases phlegmasia is the symptom which attracts attention. The onset of phlebitis in these latter cases is most insidious. The phlegmasia will appear suddenly and without the usual premonitory warnings from the patient. If it be watched for by daily examination of the limb it may be detected in its early subacute phase and before it gives rise to the symptoms of complete obliteration; the explanation being that in these tuberculous cases phlebitis remains longer in its non-obliterating or parietal stage. In these patients the latent form of the disease may often be met with and in them the non-obliterating form of the complaint may be most easily observed.

Leaving aside these, which may be considered exceptional phe-

nomena, the variety of phlebitis most commonly met with in tuberculosis is the subacute form. The patient complains of vague pains in the lower limb, pressure on the muscular masses in the calf is painful, sometimes the pain is marked on the inside of the thigh, about the level of Hunter's canal, and below Poupart's ligament at the confluence of the saphena and femoral veins. Œdema of the foot makes its appearance, at first slight in degree, and slowly mounts the limb. Then some of the superficial veins become dilated and the picture is complete. This condition remains *in statu quo* until death. The patient may complain of vague diffuse pains in the limb, but of a character very different from the ordinary painful symptoms of phlebitis. It is essentially a latent form of the disease. At the autopsy the tibial or the popliteal or even the femoral veins may be found obliterated, with more recent and partial clots in neighboring veins. Collateral circulation is established concurrently with the obstruction. Sometimes, however, the obliteration is more sudden and the consequent pains and œdematous swelling are more marked.

Finally there is a form of phlebitis met with in tuberculosis which has been called chronic phlebitis. It consists in a sclerosed condition of the veins not unlike arterial sclerosis.

Cancer and other tumors also give rise to phlebitis and phlegmasia. In these cases the phlebitis is of the latent and subacute form described above as occurring in tuberculosis.

Phlebitis of Infectious Disease.

Definition.—Inflammation of the veins arising in the course of infectious diseases.

This is all that can be positively asserted about the relation which phlebitis bears to the original disease. Proof is wanting of its being another result in the system of the action of the specific poison whose general effect gives the name to the original disease. But on the other hand it has never been proved that it is due to any diverse or even secondary infection. For the purposes of clinical comprehension it is best to consider it as *a specific inflammation arising in the course of infectious diseases and caused by the same morbid agency as the general disease in which it is encountered.* In most infectious diseases it is possible to meet with phlebitis, but it is only necessary to select a few of these for clinical study.

Typhoid Fever.—Phlegmasia is the outward sign of the venous mischief in this disease. It generally shows itself when the patient is convalescent, occurring in most cases at some period between the twenty-fifth and thirty-fifth day; or in cases of a mild or aborted character from the fourteenth to the eighteenth day.

Symptoms.—For three or four days before the actual appearance of the phlegmasia the patient experiences the premonitory pains, feelings of weight, and sensations of pins and needles in the limb affected. There is a temporary elevation of the temperature at the time when the swelling shows itself. Without exception it is the lower limbs which are the seat of the lesion, and in eighty per cent. of the cases it is the veins of the left limb which are affected. It affects both the superficial and the deeper veins. The inflammation undergoes rapid evolution, quickly producing obliteration of important veins. It spreads upward and sometimes reaches the pelvic veins. Another result of this rapid evolution of the disease is that the phlegmasia is often of short duration. After this has passed away, however, there is left œdema of a persistent and troublesome character, often dilatations and varices more or less permanent, and sometimes painful neuralgic conditions.

There is less risk of embolism in this form of phlebitis and it may on the whole be regarded as a mild form of the disease, at least so far as the resulting phlegmasia alba dolens is concerned. There seems to be no relation between the severity of the typhoid fever and the extent or acuteness of the phlebitis. Mild cases of fever may be followed with severe and painful phlegmasia, while most virulent attacks of typhoid may be altogether lacking in venous complications.

Treatment.—Rest, as in other forms of this disease, is essential. A very convenient method of securing this with comfort to the patient is to suspend the limb in a kind of canvas trough well lined with cotton wool. When it is wished to foment the limb it is only necessary to draw a piece of waterproof cloth under it in order to prevent the wool getting wet. No special medicinal treatment is required, but stimulants should be avoided as far as the nature of the general affection will permit.

Influenza.—This form of phlebitis is only too often met with in epidemics of influenza. It is sometimes, though only rarely, met with in sporadic cases—a fact which would seem to show that a certain amount of virulence in the poison is a necessary factor. In the severer cases which occur in epidemics the phlebitis appears just when the temperature tends to become normal. Other complications such as otitis, endocarditis, or nephritis may have preceded it. The pulmonary complications seem to be those which are particularly favorable to the production of phlebitis. Bronchitis, broncho-pneumonia, and pneumonia appear most prone to cause this further complication in influenza. In these cases it is when convalescence has set in that phlebitis appears. It more often attacks the lower limbs and undergoes rapid evolution. The pain is severe but disappears comparatively soon.

The œdema is marked and rapidly mounts the limb, all the indications pointing to an early venous obliteration. The inflammation has little tendency to affect both limbs; when it does so, the second outbreak is benign in character. When once the veins have become affected, the disease seems to lose its virulence; both the general and local symptoms seem to be checked. Very rarely is this phlebitis followed by fatal morbid phenomena. If the influenza be mild, as in sporadic cases, the phlebitis, when it occurs, is also mild. In such cases it generally attacks the veins of the calf and the saphena vein and disappears comparatively quickly.

One peculiarity of the phlebitis of influenza is its proneness to spread in the organism in patients already the subjects of other debilitating diseases, thus showing its septic nature. Cancerous cachectics, chlorotics, and consumptives exhibit this peculiarity of the disease.

Influenza is also specially prone to re-awaken neuralgic pains suffered in times past by patients who have had some other form of inflammation of the veins. In influenza the pains recur with pronounced neuralgic character. But this may be the extent of the venous mischief, not amounting to true phlebitis, for the symptoms disappear as soon as the influenza is cured.

In general the large veins are those which are attacked in influenza and consequently the pathological sequelæ are often persistent and troublesome. They are rarely fatal, and embolism is the exception; the reason of this is that, when the large veins are thus attacked, more care and attention are bestowed on the cases, as the symptoms are more grave.

Pneumonia.—The phlebitis occurring as a complication of pneumonia presents similar characters to that met with in influenza and therefore does not require separate notice.

Treatment.—There is nothing to add, as regards the treatment of the phlebitis either of influenza or of pneumonia, to that which has been fully stated in the description of the treatment of other forms of the disease.

Syphilis.—Phlebitis is sometimes met with in cases where there has been undoubted primary syphilis. It makes its appearance generally within three or four months of the development of the chancre. It attacks the different venous trunks and plexuses successively, and is rather chronic than acute in its character. It causes severe pain with nocturnal exacerbations. It is not accompanied with much œdema. It is regarded as a chronic form of phlebitis.

Treatment.—Mercury and iodide of potassium should be freely given. Locally, rest should be procured where possible. This is obtained by relieving the muscles of the weight of the affected part,

by means of slings and troughs of canvas lined with cotton wool, and so arranged as to allow the insertion of waterproof sheeting when use is made of moist or wet applications such as fomentations. Mercurial ointment may be applied, spread on strips of lint which are gently laid along the course of the affected veins.

Malaria.—As in syphilis, the phlebitis which is observed in cases of malaria assumes a chronic form and attacks different parts of the system either successively or simultaneously. Like the phlebitis of syphilis it is only overcome with difficulty and its effects are persistent. Patients affected long anteriorly with malarial poison present chronic indurations of the superficial veins. These indurations are painful and subject to paroxysmal attacks of pain still more severe.

Treatment.—As in syphilis, the treatment must be constitutional as well as local. The remedies in use for malarial poisoning must be pushed. Arsenic is valuable in these cases. The pain must be met with opium or its derivative morphine. Locally, the rest so essential in all cases of phlebitis must be carefully secured.

Angioma.

Definition.—A vascular tumor caused either by the enlargement of one or more vessels or by new formation of blood-vessels. Varieties: I. Aneurysm, varix, and their modifications; II. Simple angioma; III. Cavernous angioma.

The varieties in the first category do not require description here. They are fully described under their respective heads (see Aneurysm, Varix, etc.).

Simple Angioma.

Definition.—A vascular tumor in which the blood-vessels, usually capillary, are simply dilated and tortuous and held together by connective and fatty tissue; they are usually small, violet or red, slightly elevated masses, situated on the skin of the face, neck, or other part of the body, and often of congenital origin. The various forms of nævus and hemorrhoids belong to this group.

Pathological Appearances.—These angiomata are of comparatively common occurrence in glioma, but are not rarely met with in the brain, kidney, spleen, uterus, muscles, bones, hollow viscera, mammæ, and at the anal orifice. To the naked eye the tumors appear as bright red or livid patches surrounded by a number of similar spots which are not raised from the surface. In the hemorrhoidal form the dilatation of the vessels is not so marked as in other forms of simple angioma, but the increase of the tissue around the vessels is more pronounced. The walls of the small dilated veins of hemor-

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rhoids are thickened and supported by an increased quantity of connective tissue.

Beyond the appearance which they present, nævi give rise to no particular symptoms. They may be situated at different depths in the skin, or they may be subcutaneous; they are also described as capillary and venous or cavernous. The cutaneous capillary nævi are sometimes liable to ulceration of an unhealthy character on the surface. The venous or cavernous nævi are usually prominent and bulky. On pressure they may be emptied, leaving a doughy-feeling mass, which immediately refills when the pressure is removed.

Treatment.—As regards the treatment of the form of simple angioma known as nævus it must first be considered whether the case should be left to nature or whether recourse should be had to operative or other measures. If they be small and superficial and not causing much disfigurement they are best left alone, as in many cases they entirely disappear in the course of time or they may shrink or shrivel and leave mole-like marks. These terminations may sometimes be encouraged and aided by the use of iodine or liquor plumbi subacetatis. In other cases the angiomata are too extensive to be dealt with in this way. But if the nævus be large, if it be subcutaneous, if it increase in size, or if it be situated so as to occasion disfigurement, means must be adopted for its removal by operative procedure. Operations for the removal of nævi must be conducted according to one of six principles: 1. To excite inflammation in them so as to produce plugging and obliteration of the vascular tissue of which they are composed; 2. To destroy the growth by caustics; 3. To remove it by the cautery; 4. To consolidate it by electrolysis; 5. To remove it by the knife; 6. To remove it by ligature (Erichsen). In choosing between these different forms of treatment, the practitioner must be guided by the size, situation, etc., of the tumor. Small nævi can be dealt with after either of the first three plans. Care must, however, be taken if (1) be adopted not to inject strong styptics in nævi situated on the head, as the coagulation excited has been known to spread rapidly into the vessels of the brain, causing fatal results. Electrolysis (4) should be adopted in the case of deep-seated tumors. Excision with the knife (5) should be used if the nævus is capillary or venous and so situated as to render the operation practicable. Ligature (6) is the most generally adopted method and is applicable to nævi in almost all situations. There is, moreover, no risk of hemorrhage. There are several different kinds of ligature recommended and a practical acquaintance with these is advantageous; but the shape, position, and size of the tumor must be the factors which determine the kind to be used.

Hemorrhoids.

Definition.—Angiomata situated near the anus and consisting essentially of thickened and dilated vessels, chiefly veins. Piles may be external or internal, the external protruding through the anus, the internal being situated within the gut; an intermediate variety has been called intero-external. They are also called open or blind, the former term being used when they bleed, the latter when they do not. External piles are always blind. Internal piles sometimes bleed.

Symptoms.—External piles are nodular masses of bluish-white color and firm consistence. They may be sessile or pedunculate, single or multiple. They are not painful when quiescent but exquisitely so when inflamed. They are covered with thickened skin.

Internal piles are situated inside the anus, they are covered with mucous membrane, and they bleed readily, indeed hemorrhage is their earliest symptom. As they grow they are protruded externally in the act of defecation or in walking, and by the constriction of the sphincter become painful until they are returned. They give rise to backache which extends to the thighs, to mucous discharge from the anus, and to bladder troubles. When inflamed they may cause much constitutional disturbance. With time their protuberance becomes permanent and they are then generally surrounded by a ring of external piles.

Causes.—These are predisposing and exciting. Among the *predisposing causes* there is one element which cannot be overlooked, and this is the peculiar anatomico-pathological arrangement of the veins of the rectum. This will be definitely described under the heading of Pathology, but it is necessary to call attention to it here, as the other predisposing causes are all more or less connected with this arrangement, that is, they produce hemorrhoids by their influence thereon. These causes are age, sex, sedentary life, and alcoholic and other excesses.

In young men from eighteen to twenty years of age, especially in those of a phlegmatic temperament and languid circulation, piles are frequently met with. After this period and on to middle life their frequency diminishes and from then again becomes more frequent.

The affection is more common in men than women in early life, but after that it occurs with equal frequency in the two sexes. Women are comparatively free during early life because of the menstrual flow which relieves the pressure of the blood. But during pregnancy the pressure of the gravid uterus renders them particularly prone to the trouble. Also after the climacteric they are subject to congestions which retard the portal circulation and so tend to produce piles.

The abnormalities of the liver and of the portal circulation so

commonly produced in *sedentary life* are powerful factors in the production of piles. Alcoholic excess also acts by its influence in the liver.

Exciting Causes.—The chief of these are local irritation such as hard riding, the habitual use of powerful purgative medicines such as aloes, and the existence of other diseases about the rectum such as fistula, ulcer, or stricture. Uterine affections and genito-urinary disorders may give rise to hemorrhoids.

Complications.—The most common are backache, ulcerations of the mucous membrane of the rectum giving rise to a mucous discharge, suppuration, fissure, fistula, and prolapse of the rectum.

Pathology.—The hemorrhoidal veins discharge their contents in two directions: the greater part of the blood by means of the inferior mesenteric veins reaches the portal system, while the remainder passes into the internal iliac vein through branches that accompany the middle and inferior hemorrhoidal arteries. In these arrangements we see the elements which predispose to congestion and consequent dilatation of the vessels of the part. There is the large and intricate plexus of veins in which, as in all similar networks, there is a tendency for the blood to circulate slowly; the natural tendency to stasis is also much increased by the dependent position of the part and by the absence of the valves in the superior hemorrhoidal veins and the vessels into which they pour their contents, and thus the whole pressure of the column of blood in the portal system is brought to bear upon the hemorrhoidal plexus (Erichsen). Any obstruction, either in the liver itself or elsewhere in the course of these veins, will therefore bring about dilatation at the point of greatest stress, and this will be the hemorrhoidal plexus. Again, not only are these veins called upon to support a heavy column of blood, but they receive scanty support from surrounding structures. They are situated between a muscular layer on the one side and a yielding areolar and soft mucous membrane on the other. Consequently, constricted as they are during the act of defecation, they naturally tend to give way in the line of least resistance and this is toward the mucous surface. The mucous membrane itself tends to prolapse during defecation and is therefore easily pushed in front of the protruding and growing angioma; or if the yielding vessel or vessels be situated below the sphincter, the external skin instead of mucous membrane will be pushed out. In the early stage, the pile, whether it be external or internal, consists essentially of a varicose portion of the hemorrhoidal plexus or of the small veins which discharge their contents into it. It is quite soft and compressible and when cut into will be found to consist of one or more sacculated venous sinuses surrounded by areolar tissue. The next stage is one of inflammation, affecting perhaps only the stretched

vein walls, such as occurs in varix; it is an inflammation which is more or less compensatory in its effects, causing a thickening and stiffening of the vein wall; it may lead to coagulation of the contained blood, and, if more extensive, will cause an hypertrophy of the external areolar or connective tissue. Microscopically it will be seen that the primary phlebitis has resulted in a thickening and strengthening of the adventitia. External piles, when cut into in this stage, often present an appearance of increased connective tissue permeated with numerous small vessels with thickened walls. But internal piles are more distinctly dilated venous sacs surrounded by more or less areolar tissue, and under the microscope their endothelial lining can be easily demonstrated.

Treatment.—It will be evident from the above that the treatment of hemorrhoids, to be efficient, must be no mere local tinkering. It must deal also with the cause of the disease. In other words the treatment must be both constitutional and local.

Constitutional treatment must of course vary in accordance with the original cause of the malady and with the temperament of the patient. In all cases the irritation of the piles themselves must be diminished by the use of aperient medicines, so that the motions may be softened as much as possible, short of violent purgation. Such aperients are found among the milder purgative medicines that tend to excite a free secretion from the mucous membrane of the intestines and to relieve congestion of the portal system. Castor oil, confection of senna, confection of sulphur are all beneficial. They should be taken regularly in small doses. Generally it will be found sufficient to administer them two or three times in the week. A very useful mixture is composed of equal parts of the confection of sulphur and bitartrate of potash with two parts of confection of senna and a little syrup. Two drachms of this may be taken every other night. Laxative mineral waters such as Hunyadi János, Friedrichshall, Æsculap, or Vichy, taken every morning, are also useful.

Tincture of hamamelis materially relieves and sometimes cures bleeding piles. It may be given both by the mouth and as an enema. For the same purpose, *i.e.*, of softening the motions, enemata of soap and water may be used, cold if the patient is of relaxed and debilitated habit of body, and tepid if he is plethoric. An injection of cold water after a motion will often relieve the irritation which it causes.

Local Treatment.—This may be either palliative or operative. As *palliative* measures, sponging the part with cold water, and astringent injections for internal piles are useful. For the latter purpose we may use perchloride of iron or a combination of one grain of sulphate of iron with ten drops of the perchloride to an ounce of water; two ounces of this should be injected every night and left in the rec-

tum. Unguentum gallæ cum opio and unguentum hamamelis are also useful local applications. Leeches are sometimes used for the relief of inflamed external piles. Hot fomentations are also beneficially employed in this condition.

Operative Treatment.—Sir E. Erichsen writes: "The means indicated above (as abstracted from his work) are usually sufficient in ordinary cases of piles, but if the disease attain an inconvenient size, giving rise to general irritation and local uneasiness, or if the abundance of the hemorrhage be such as seriously to interfere with the health of the patient, it will become necessary at last to have recourse to operative interference with a view of removing the diseased structure. No operation should ever be undertaken while the pile is in an inflamed state, lest unhealthy inflammation be set up in the part; it is also well to get the patient's health into a good state, as erysipelas may otherwise follow the operation; and before proceeding to perform it, care should be taken that the bowels are well opened by a purgative and the rectum cleaned by an enema of warm water. If the patient is suffering from cirrhosis of the liver no operation is justifiable.

"There are five plans of treatment commonly adopted for the removal of piles: viz., excision of the tumor, its strangulation by ligature, its destruction by clamp and cautery, by crushing, and by the use of caustics." ("Practice and Art of Surgery," Vol. II.)

There is also another method of treatment of external and protruding piles which has been strongly recommended. This consists in the injection, through a large-sized hollow needle, into the tumor itself of a mixture of one-third to one-half pure carbolic acid and one-half to two-thirds glycerin. This causes the death and destruction of the hemorrhoidal tumor, which is eventually cast off. The method is comparatively painless.

Cavernous Angioma.

Definition.—A vascular tumor, in which, besides the arteries, veins, and capillaries of simple angioma, there are connected with the vessels alveolar spaces communicating freely with each other, lined with an endothelium consisting of flat scales. The intervening structure consists of connective tissue with occasional unstriped muscular fibre. It is spongy to the feel, bluish in color, and sometimes pulsating.

Locality.—Cavernous angiomata are most commonly met with in the skin and subcutaneous connective tissue, especially near a mucous orifice; but they may also occur in the orbit, liver, spleen, kidney, or muscles. Vascular tumors of the urethra belong to this category. They are more common in women than in men. Their origin is generally gonorrhœal. They are excessively painful, particularly after micturition, and bleed freely.

Treatment.—Vascular tumors of the urethra must be removed and this may be done in one of four different ways, namely, by excision ligature, caustics, or the actual cautery. The other tumors of this variety must be dealt with as described under nævi, and the plan adopted must depend upon their position and the amount of inconvenience to which they give rise.

Several other varieties of angiomata have been described, such as lipogenous, phlebogenous, racemose, etc.; but the above-described varieties really include all these.

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DISEASES OF THE LYMPHATIC VESSELS.

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DISEASES OF THE LYMPHATIC VESSELS.

LYMPHANGITIS OR ANGIOLEUCITIS.

Inflammation of the lymphatic vessels is almost always accompanied by a like condition of the neighboring lymphatic glands, and the above terms denote accordingly a predominant rather than an exclusive implication of the vessels.

Etiology.

The direct causes may be set forth as follows:

1. Injuries and contusions without breach of the surface of the skin. Such slight causes are more likely to operate on a part whose nutrition is deficient or whose circulation is sluggish; they determine the attacks of lymphangitis which occur in the course of elephantiasis. Lymphangitis produced by the heat of the sun is included under this heading.

2. Introduction of some septic product through a breach of the surface. Such a breach may be an ulcer or open wound, or on the other hand be invisible to the naked eye, as is often the case in post-mortem poisoning.

3. Extension from inflammation in the immediate vicinity, *e.g.*, abscesses, phlebitis, etc.

4. Lymphangitis incidental to the course of specific diseases, such as scarlet fever, measles, diphtheria, tubercle, syphilis, and gonorrhœa; in these, inflammation of glands is the more prominent feature.

Lymphangitis is more easily set up in young subjects than in those of middle and advanced age. A predisposition is established by alcoholism, chronic gout, and renal disease, and the affection is more likely to occur in those who are debilitated from any cause than in those who are robust and strong.

Pathology.

The external coat of the vessel becomes swollen and thick, and the inflammation usually extends into the surrounding connective tissue leading to exudation and thickening.

The intima of the vessel is uneven and opaque and its endothelium undergoes degeneration.

The contents of the vessel become cloudy and frequently coagulate. If circumstances are favorable, and the inflammation subsides, there is complete resolution; the thrombus within and the exudation without the vessel become absorbed and, but for a temporary dilatation, the latter returns to its normal state. In other instances the exudation without the vessel becomes organized and later sclerosed, the vessel lumen is obliterated and some degree of permanent thickening results. In yet other and unfavorable cases the coagulum within the vessel breaks down, the suppuration extends to the vessel wall and the exudation outside it, and an abscess forms, leading sometimes to extensive cellulitis.

Symptoms.

Pain is always present and varies, like the other symptoms, with the severity and extent of the process; it is increased by movement and is accompanied by a feeling of tension in the part. When suppuration threatens, pain increases in severity and becomes throbbing in character and there is tenderness on pressure, especially along the lines of the affected vessels as far as, and including, the next chain of glands.

Objectively the inflammation presents two forms: One—*reticular lymphangitis*—in which the smaller capillary vessels are involved, and the skin presents a diffused pink blush; and the other—*tubular lymphangitis*—in which the larger vessels are inflamed. These latter appear on the surface as straight or wavy red lines travelling toward the neighboring glands and, owing to the infiltration in and around them, are perceptible to the touch as firm knotty cords. If the inflammation is extensive there may be a considerable lymphatic œdema and the circumference of a limb may be much increased.

The constitutional symptoms will vary according to the extent of the local inflammation, the severity of the cause, and the general health and resisting power of the patient. There will be a varying degree of fever, with the usual accompaniments of malaise, thirst, and anorexia. Where suppuration supervenes or where some virulent septic poison is the cause of the illness, high and irregular pyrexia

with rigors, sweating, rapid wasting, and other symptoms of a septicæmic character may make their appearance, and the case will assume a grave aspect.

Prognosis.

But for the contingency of suppuration and septic intoxication, the prognosis is favorable. The general health and robustness of the patient have a marked influence. Thus recovery is slow in the subjects of alcoholism, chronic gout, and renal mischief, and in those debilitated by poor living and overwork.

Treatment.

The first indication is to remove the cause, if possible. If such be an unhealthy wound it should be rendered clean and healthy, and if there be a collection of pus a free vent should be at once given to it.

The affected part should, as far as possible, be afforded complete rest and, if it be a limb, it should be raised.

The local application of evaporating lead and spirit lotion affords relief and checks the inflammation; in other cases, and these often the more severe ones, heat and moisture in the form of fomentations, with or without the addition of belladonna, give better results.

If any collections of pus form they should be freely incised. For constitutional treatment the diet should be a nourishing one; solid food need not be withheld unless a high degree of fever causes it to disagree. The bowels should be kept freely open. Quinine, and later iron in addition, are the most efficient medicines. Stimulants will be needed only in severe cases. Very often a few days' residence by the sea will bring about a rapid cure where other means have been inefficient.

The lymphangitis which occurs in the course of constitutional diseases does not usually require separate treatment.

In chronic conditions affecting the lymph glands and vessels, such as tubercle and carcinoma, the changes are mainly in the former; those in the vessels are of less importance and need not detain us here.

OBSTRUCTION OF THE THORACIC DUCT OR ITS VENOUS OUTLET.

Two groups of consequences may result from this condition. In both there are lymph stasis and increased lymph pressure on the distal side of the obstacle; in one a lymph varix and establishment

of a collateral circulation follow, and in the other rupture of the thoracic duct.

1. Let us suppose the thoracic duct to be obstructed at its upper termination. The first effect of the blockage will be a stasis of lymph throughout all the lymphatics draining into the thoracic duct, with a considerable rise of pressure. On the other hand, the pressure in those lymphatics draining into the right lymphatic trunk will remain as before. It will follow that where the former system inosculates with the latter, there will be a flow of lymph from the points of higher toward those of lower pressure.

To recall briefly the anatomical facts: The lymphatics empty their contents into the venous system by two vessels, the left and right lymphatic trunks. The left lymphatic trunk, or thoracic duct, commences in the abdomen at the receptaculum chyli, is about eighteen inches long, and terminates at the junction of the left subclavian and internal jugular veins. It receives lymph from the left side of the head and neck, from the left arm, left side of the thorax, the whole of the trunk below the thorax, except the upper part of the right half of the anterior abdominal wall, from the legs, and from all viscera except the convex surface of the liver. The right lymphatic trunk drains the right side of the head and neck, the right arm, the right thorax, a small portion of the right upper abdomen, and the convex surface of the liver; it is less than an inch long and enters the venous system at the junction of the right jugular and subclavian veins.

If from any cause the thoracic duct became obstructed high up, it would seem probable that the raised pressure within its system would lead to the gradual formation of new channels along the anastomoses between its system and that of the right lymphatic trunk. In this way a collateral circulation would be established and at the same time the lymph stream would be considerably diverted. Thus the lymph from the legs, instead of draining into the lumbar and iliac from the inguinal glands, would probably find its line of least resistance along the lymphatics of the abdominal wall and their enlarged anastomoses with the right lymphatic trunk system; similarly one would expect the visceral lymphatics, through their communications with the deep and superficial parietal lymphatics, to empty their contents into the right trunk system. The lymphatics of the bladder, for instance, drain largely into the internal iliac glands, which communicate with the external iliac and lateral lumbar glands, these two latter connecting in turn with the lymphatics of the abdominal wall.

Applying the same line of reasoning, the chyle unable to dis-

charge itself into the thoracic duct would, as soon as the collateral circulation between the two systems was established, regurgitate along the visceral lymphatics, thence pass probably into the parietal lymphatics and so into the blood circulation. Following then an obstruction of the thoracic duct, the course of the lymph stream would be materially altered and in the more immediate tributaries of the duct actually reversed. If, instead of being at the upper extremity, the obstruction is situated lower down the duct, the lymph will reach the blood stream not only by anastomoses with the right trunk system but also by communications between the vessels which drain into the thoracic duct above and below the occlusion. With the complete establishment of the collateral circulation the excessive pressure in the thoracic duct system will be abated, but, by the time this adaptation has been established, the stasis and increased pressure will have left permanent marks on the lymphatic vessels and the latter will have become tortuous, dilated, and varicose.

We can find an interesting parallel to the above condition in the venous system. If the two innominate veins be obstructed by a mediastinal tumor, there are impediments to outflow and back pressure, with establishment of a collateral circulation through the superficial veins of the chest and back, and through anastomoses between these and the veins of the lower half of the trunk. All the superficial veins involved in this collateral circulation become enlarged and those of the chest and back, in which the natural direction of the current is reversed, become very markedly tortuous and varicose. It is well known that veins become varicose when the circulation in them is reversed, and this fact is no doubt due to the presence of the valves. Another comparison is to be found in obstruction of the portal vein, the result, say, of hepatic cirrhosis. This similarly causes increased internal pressure, venous varix, and even hemorrhage.

A consideration of the structure and nature of the lymph ways and channels would lead us to expect that the effects of back pressure due to the blocking of the main outfall would be even more marked in their case than we have seen them to be in that of the veins.

Unlike the arteries and veins, the lymphatics do not constitute a closed system of tubes; they commence as mere spaces without definite walls, and even in the lymph capillaries and larger lymphatic vessels, where the channels are definitely tubular, the walls are more slender and yielding than those of the corresponding veins. The lymphatics further are richly provided with valves which strongly favor the formation of varices when the direction of the circulation is reversed.

The lymph varices thus formed may be numerous and diffused in

their distribution, or they may be aggregated together so as to form lymphangiomata. Further the walls of these lymph varices and lymphangiomata are liable from time to time to become thinned and to rupture. In this way lymph or chyle escapes, either by transudation or through a definitely formed fistula, and we get a condition known as lymphorrhœa. It has been usual to apply this latter term only to lymphatic fistulæ on the surface of the body, but there is no sufficient reason for the limitation.

If escape of chyle or lymph takes place, the resulting condition varies according to the situation of the lymph varix or lymphangioma. If the latter leak into the peritoneal cavity, we get chylous ascites, and if into the pleural cavity, chylothorax; if it lies in relation to the bladder, we get the condition known clinically as chyluria; if it is situated in the tunica vaginalis, we get chylocele; if beneath the mucous membrane of the intestine, chylous diarrhœa; and if in the skin or subcutaneous tissue, vesicles or pustules discharging chyle or lymph (external lymphorrhœa). As regards chyluria this view of its causation was first suggested by Carter as far back as 1862 (Medico-Chirurgical Transactions, London, XLV., p. 189). In no other way can the presence of chyle in, say, the urine or in discharges from cutaneous fistulæ be adequately explained. For chyle to reach the lymphatics around the bladder or in the skin there must be regurgitation in the thoracic duct or its immediate tributaries and we cannot suppose that such regurgitation could take place if the duct and its venous outlet were unobstructed.

A certain amount of effusion of chyle and lymph could be, and doubtless is, explained by the process of transudation under back pressure, as in the case of obstructed veins, without the supposition of lymphatic fistulæ being formed, but, apart from the fact that such fistulæ can be demonstrated, the effusions of chyle are too local and too profuse to be adequately accounted for without them.

It is a fact further, as will be seen below when these morbid conditions are individually considered, that these discharges of chyle are often spasmodic in their appearances, the intermissions varying from a few hours to many months or even years. This fact would show that definite though unusual channels exist for the carrying away of the chyle during the intermission, unless the obstruction to the thoracic duct is itself of an intermittent character, and the latter explanation in no way receives support from the few post-mortem examinations that have been made in these cases.

But the question may arise: Is the fluid discharged in chylous ascites, chyluria, etc., true chyle? And this question suggests a brief comparative examination of their properties.

To the naked eye the resemblance is close. Chylous effusion may so closely resemble milk as for the moment to be mistaken for it, if two vessels, one containing milk and the other the effusion, be placed side by side. Under the microscope the fat is seen to be in the same fine molecular state that we are familiar with in chyle. Qualitatively the chemical compositions of chyle and chylous effusions closely agree. Quantitatively there are wider divergencies, it is true, but these are not more marked in the cases of the effusions than among different analyses of chyle itself. Thus taking two analyses of human chyle quoted by Halliburton ("Text-book of Chemical Physiology," p. 336) we find the fat varies from 0.92 per cent. in one to 2.7 per cent. in the other, and the albumin from 7.1 to 1.1 per cent.

We have further, in some cases of effusions, to allow for the admixture with the chyle of other fluids more properly belonging to the situations invaded by the former. For this reason the percentages of both albumin and fat in chylous urine are much lower than those of chyle, whereas in chylous ascites and chylous skin discharge the differences are slight.

The following table, based on many analyses, enables comparisons to be made between the amounts of fat and albumin contained in chyle and the various effusions:

Albumin.	Chyle.	Chylous Ascites.	Chyluria.	Chylous Skin Discharge.
Maximum	7.1	4.46	1.4	5.7
Minimum	1.1	1.9	0.12	1.7
Average	4.1	3.18	0.76	3.7
Fat.				
Maximum	2.7	1.78	1.39	3.06
Minimum	0.92	0.93	0.06	0.03
Average	1.8	1.35	0.72	1.8

As showing the chylous origin of these effusions, some observers have noted a relation between the amount of fat given as food and the percentage of fat in the effusion. Hensen observed this in a case of lymph scrotum (Pflüger's *Archiv*, X., p. 94) and Strauss in chylous ascites.

The presence of sugar in some chylous effusions is of interest and may here be considered. Analyses show the amount of sugar in chyle to be very small and its absorption is held by physiologists to be effected not by the lacteals but by the blood, although Ginsberg (Pflüger's *Archiv*, XLIV., p. 306) does record the fact that increasing the sugar in the food increases the amount in the chyle. The compositions of the majority of chylous effusions support this view and show no more than traces of sugar; on the other hand sugar in

appreciable and quantitatively analyzable amounts has been recorded in a minority of cases large enough to demand attention.

Thus in Whitla's case of chylous ascites (see p. 657), in which the chyle escaped through an afterwards demonstrated aperture in the thoracic duct, 0.21 per cent. of sugar was found by Professor Hay.

Again in cases of chylous ascites recorded by Quincke, Bargebuhr, and others, the effusion filtrate after the separation of the fat and albumin reduced cupric oxide and therefore, unless the reduction was effected by some other agents, we must infer that sugar was present in appreciable quantities.

The same may be said of chylous urines that, although most of them do not contain sugar, yet there have been several quite definite instances in which the contrary was the case. Among others may be mentioned a case by Morrison (Transactions of the Pathological Society, XXIX., 394). It is difficult to explain the presence of sugar in these effusions except by supposing for the nonce its presence in the chyle. Professor Hay regarded his discovery of sugar in chylous ascitic fluid as definite evidence of its presence in chyle, and both Bargebuhr and Senator went further and regarded the presence of sugar in an effusion, provided diabetes was absent, as a criterion of such effusion being chylous. This latter conclusion seems a little confident in face of the absence of appreciable quantities of sugar in analyses of chyle obtained pure from the thoracic duct and not admixed with other fluids.

Nearly all chylous effusions contain traces of blood and sometimes have a pinkish hue, due to this cause, but, except in a few cases of chyluria in which there is concurrent hæmaturia, the amount of blood is quite small. Its presence receives adequate explanation when it is remembered that specimens of lymph taken from the spleen and thoracic duct have been found to contain blood elements and that stasis in the larger lymphatics, produced by ligature of the thoracic duct, is followed by an increase of these blood elements, probably the result of progressive changes in the lymph.

Pathological Anatomy.

It will now be of interest to see how far the foregoing views receive support from actual post-mortem observations and to what extent chylous effusions, lymphatic varices, and tumors are associated with obstruction of the thoracic duct or its venous outlet.

Chyluria.—It will be convenient to consider first the anatomy of cases of chylous urine. In a case of fatal chyluria observed and recorded with great completeness by Dr. Stephen Mackenzie (Trans-

actions of the Pathological Society, London, XXXIII., p. 394), an elaborate dissection was made from behind of the thoracic duct and its tributary vessels.

The whole of the space between the kidneys was occupied by a mass of dilated and varicose lymphatics, which extended downwards along the course of the iliac arteries. The tumor included the lumbar, iliac, and renal groups of lymphatics, and varix was especially marked on the left side. The thoracic duct was thus described: "The receptaculum chyli commences by two large lymph sinuses about the size of a pencil, one from each side of the aorta, and is joined opposite the aortic opening of the diaphragm by a third large sinus, about the same size. The duct now ascends sinuous and much pouched for three or four inches, varying in diameter from three-eighths to one-half inch, pervious for the first one and a half inches above the aortic opening of the diaphragm, then filled with loose clot for one and a half inches, after which it is lost in a tough thick mass (inflammatory?). About four inches above this point, when it can again be traced, although still involved in dense tissue, it is now the size of a crow quill, impervious, and tending to the left side behind the aorta. At this termination in the angle between the left subclavian and internal jugular veins, it passes through a mass of lymphatic tissue, is pervious, and about the size of a goose quill." Although the varix closely embraced the urinary tracts, no fistulous communication was observed. The chylous condition of urine had, however, in large degree abated before the patient's death.

Havelburg (*Archiv für Pathologische Anatomie*, LXXXIX., p. 365, 1882) has published a fatal case of chyluria in which there was a partial autopsy.

The patient was a woman, aged thirty-two, who had lived many years in Brazil, and her illness began with abdominal pain chiefly on the left side. After this symptom had persisted for some time she suddenly felt something give way in the abdomen, and from that time pain ceased and chyluria appeared. After the illness had endured a considerable time, the patient was seized with vomiting and diarrhœa accompanied by fever. She passed into a typhoid state and death followed.

Post mortem a large sac-like tumor was found extending from the left side of the true pelvis below to the upper border of the left kidney above. The tumor consisted of many loculi and its contents were chylous. Within its substance could be felt many nodules as large as walnuts, which were without doubt swollen lymph glands. The left and upper wall of the bladder lay embedded in the tumor, and when the bladder was opened a fistula was discovered through which milky fluid could be pressed. In the corresponding part of the right side of the abdomen no similar tumor was observed. The lymphatics of the mesentery were much enlarged and felt like thickened cords.

Unfortunately, in consequence of the autopsy having to be limited to the abdomen, no examination of the thoracic duct could be made,

and so this valuable observation lacks completeness. The concurrent enlargement of the lymphatics of the mesentery, however, strongly points to an obstruction at some point above the junction of the intestinal with the lumbar and iliac vessels, that is, at or above the receptaculum chyli. Although a report of the microscopic structure of the tumor is wanting, the description suggests its inclusion under the cavernous or mixed cavernous and cystic lymph tumors—lymphangioma cavernosum cysticum.

Manson records (Davidson's "Hygiene and Diseases of Warm Climates," p. 814) a post-mortem examination on a patient who had suffered, like those above mentioned, from filarial chyluria.

The thoracic duct, which was examined partly fresh and partly after preservation in spirit, commenced below in an ill-defined tumor, situated in front of the vertebræ and below the diaphragm; it was enlarged throughout, though variously, its diameter being equal to the little finger below and to a goose quill above. The upper two to three inches of the duct with its venous outlet were quite occluded. Below this the duct was patent and its lumen was occupied throughout its length by a firm thrombus, which in its upper extent was closely and almost structurally connected with the wall of the duct, but below, where the latter was wider, it was loose and easily detachable. The mass in which the thoracic duct commenced, extended downwards as far as the right iliac fossa and closely embraced one of the ureters. The kidneys, ureters, and bladder were found to be quite healthy and no communication whatever could be found between them and the tumor. The latter on section was tough and fibrous and showed here and there gaping vessels with the walls cut across, which were probably dilated lymphatics.

No microscopic examination of the tumor is recorded.

There is one more case of fatal chyluria reported by Ewald (*Deutsche klinische Wochenschrift*, 1881, p. 624 and *Bericht der Naturforscher Versammlung zu Danzig*, 1880), in which the post-mortem was made by Ponfick. The notes of the autopsy are not so full as those of the preceding cases, but as far as they go they record similar changes. The thoracic duct was exceedingly dilated, its walls were thickened, and its inner surface rugose. The lymphatics of the abdomen were thick, cord-like, and turgescent, and contained bluish-red clots, and the glands were swollen in the neighborhood of the enlarged vessels.

Chylous Ascites and Chylothorax.—The association between chyluria and thoracic-duct obstruction is clearly shown by the above cases, and we will now see how far the same relation holds in the case of chylous ascites and chylothorax.

Martin (Transactions of the Pathological Society, XLII., 1891, p. 93) reports the case of a woman, aged thirty-nine, who was admitted into the hospital complaining of cough, dyspnœa, vomiting, en-

largement of the abdomen, etc. The physical signs in the abdomen were those of fluid. At first no active treatment was adopted, but five weeks after admission, as the enlargement was progressing and the lower limbs had become œdematous, paracentesis was performed and had to be repeated five days later. On the first occasion forty-eight, and on the second sixteen, ounces of typically chylous fluid were withdrawn. The patient was seized with sudden dyspnœa the night after the second tapping and died of asphyxia within an hour. Post mortem the abdomen was found to contain one hundred and eighty ounces of chylous fluid. The surfaces of the small intestine and to a less extent of the large intestine showed ramifications of dilated lymphatics containing a milky fluid. The whole of the mesentery presented a milky arborescent appearance. The left pulmonary pleura showed the same characters. The left pleural space contained ten ounces of milky fluid and the lymphatics of both lungs were seen to be distended. The thoracic duct and receptaculum chyli could not be found. The left innominate vein was small but patent except quite at its commencement where it was occluded by contraction. The left subclavian and internal jugular veins were both occluded for the last two inches and mere fibrous bands had for this length replaced them. The azygos and intercostal veins were much distended. The veins on the right side of the neck were normal.

Here then we have both abdominal and pleural chylous effusions and in this, as in the following case, the cause was probably blocking of the venous outlet.

Renvers records (*Berliner klinische Wochenschrift*, XXVII., p. 320) chylous ascites coupled with venous engorgement of the upper half of the trunk. In the left supraclavicular fossa was a tumor the size of a pigeon's egg. At the autopsy the abdomen was found to contain chylous effusion, and the thoracic duct was serpiginous, thickened, and dilated, especially above, just below the obstruction. There was extensive thrombosis of the left innominate vein; the left subclavian vein at its termination was quite obliterated, and on being traced a little way back was found to be dilated into a cyst the size of a pigeon's egg, which during life had been felt as a tumor above the clavicle. There was a history in this case of recent attacks of erysipelas prior to the fatal illness, which may have explained the thrombosis of the neck vessels.

This case is also of interest in relation to the view held by some authorities, Unna among others, that blockage of lymphatic trunks does not alone suffice to produce lymph varix, but that a concurrent venous obstruction is also needed.

The association of thrombosis of the vessels of the neck with chylous ascites is further illustrated in a communication by Ormerod (Transactions of the Pathological Society, XIX., p. 199). The left subclavian vein and its tributaries were found thrombosed, and chylous ascites was demonstrated both by paracentesis and at the autopsy.

We will now pass to two cases illustrative of lymph varix on the

surface of the body with discharge of lymph or chyle (external lymphorrhœa). In the first case obstruction of the thoracic duct was demonstrated post mortem, and the second was associated with chyluria, although later nothing abnormal was found in the larger lymphatic trunks.

The former is recorded by Bryk and quoted by Unna ("Histopathologie der Hautkrankheiten").

The patient, who was a man, aged twenty-six, showed a diffused and almost universal lymph varix with lymphorrhœa; his illness had commenced at the age of fifteen and had progressed steadily since; the legs were swollen as far as the knees, on the feet were numerous vesicles which to some extent had become confluent and which discharged lymph in abundance. The œdema of the feet was hard and elephantiasic, the inguinal glands were enlarged and stood out as tumors. On both hands were papules and vesicles, the lymph glands of both arms were enlarged, and the left upper extremity was œdematous.

At the post-mortem a glandular tumor, the size of a goose's egg and compressing the thoracic duct, was found in the posterior mediastinum. The diameter of the duct at the point of compression had been reduced to 2 mm., and from here downwards towards the inguinal region were to be seen convolutions of dilated lymphatics and enlarged lymph glands. There was a right pleural effusion and the subpleural lymphatics on the same side were dilated. The lymph vessels of the feet were dilated and varicose, with cyst-like dilatations which were partly thrombosed. The same condition was found in the left arm. The vesicles on the surface were shown to be connected with the lymphangiomatous network beneath and they showed moreover the typical endothelium of lymphatic vessels—in short they were superficial cystic dilatations of the latter. All these dilated lymphatics of the periphery could be easily injected from the central lymphatics below the obstruction.

The second case is related by Sir W. Roberts ("Urinary and Renal Diseases," p. 377). On admission to the hospital the patient's abdomen was seen to be studded with vesicles of milky contents. The integument in the neighborhood of the vesicles was dull red, thickened, and tumid. From time to time one or two vesicles discharged a lymphous or milky fluid, the amount varying from a few ounces to several pints in twenty-four hours. Sometimes the vesicles would be flaccid and at other times distended; sometimes they would be pale and at other times milky. Milkiness and fulness went with improved health and recent ingestion of food, while paleness and flaccidity followed fasting, loss of appetite, feverishness, etc.; at any one time the vesicles all presented a uniform appearance. The fluid discharged from the vesicles had the usual characters of lymph and chyle. Twice during the illness chyluria was observed. The patient died of intercurrent phthisis. Some time before death the discharge had ceased and the eruption had faded. Post mortem an examination of the affected abdominal parietes showed the skin and subcutaneous tissues to be thickened, in some places to the extent of an inch. These tissues were permeated with a network of channels

varying in diameter from a hair to a crow quill and freely communicating one with the other. These latter were clearly dilated lymphatics and the vesicles communicated with them. There was increase of the connective tissue between the dilated vessels; in short, the abdominal wall was the seat of a lymphangioma.

An interesting observation in comparative pathology may fittingly be mentioned here. Virchow (*Archiv*, VII., p. 130) demonstrated in a calf a considerable dilatation of the lymph tracts, associated with occlusion of the thoracic duct, resulting from thrombosis of the left jugular vein.

Leaving now these morbid changes found in association with chylous and lymphous effusions, we may turn for a brief space to experimental pathology. Ligature of the thoracic duct in animals has been shown to be followed by lymph varices and establishment of an anastomotic circulation, or by rupture. Thus Sir Astley Cooper ("Medical Records and Researches") tied the thoracic duct in dogs. In every instance but one the animals died promptly, and rupture of the receptaculum chyli was found. In the dog which survived, there was an efficient anastomosis with the tributaries of right lymphatic duct. Gradual compression of the thoracic duct would tend to the formation of varix, whereas sudden occlusion would be more likely to produce rupture. Sir Astley Cooper found that pressure on the thoracic duct for only a few minutes during digestion caused the receptaculum to rupture.

Dupuytren and others have shown that experimental ligation is gradually followed by the establishment of a collateral circulation.

An interesting experiment performed and recorded by Wegner (*Archiv für klinische Chirurgie* [Langenbeck], XX.) shows the effect on the lymphatics of increasing their internal pressure. He kept the abdominal cavity of a rabbit filled with air under pressure for several weeks; the air entered the lymph vessels through the stomata and found its way between the layers of the omentum and those of the meso-rectum. Under this pressure the lymphatics became enormously dilated, less so at the valves than between them, so that the vessels formed a series of cysts which resembled the beads of a rosary. If the pressure was made to persist the air cysts continued to grow and formed large tumors. Wegner figures such a formation —the size of a child's fist growing up from the pelvis into the abdomen of a rabbit (Fig. 42); it consists of numerous vesicles closely bunched together like grapes, and varying in size, the larger ones resulting from the confluence of two or more of the smaller. For the most part the vesicles communicated with one another and all had the characters of lymphatic vessels, their inter-

nal surfaces showing the typical endothelium when stained with nitrate of silver. Wegner regards such an artificially produced tumor as a true lymphangioma and holds that growths similar in their form and mode of production are found in man. In this connection the above experiment is of importance as showing that lymph varix and lymphangioma can be produced by increase of pressure within the lymph vessels.

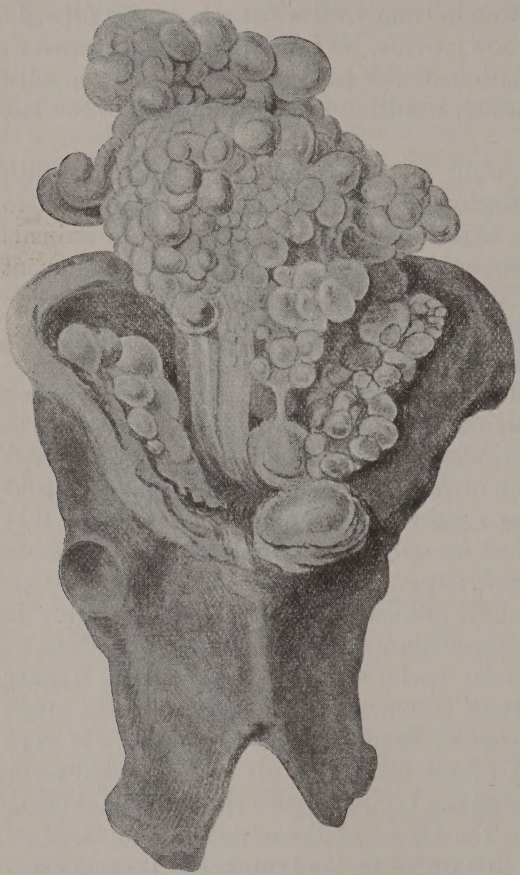

FIG. 42.—Lymphangioma Cysticum, Experimentally Produced. (After Wegner.)

In the face of the foregoing considerations, both clinical and pathological, the conclusion is on the whole irresistible that a causal relationship exists between thoracic duct-obstruction and chylous effusions, and that the sequence of events is obstructional lymph stasis, dilatation, lymph varices or lymphangiomata, local thinning of the walls of the latter, and escape of chyle either by means of transudation or visible fistula, the chyle deviating through unusual channels on its way to the blood stream by means of newly established anastomoses.

Sometimes the varicose lymph channels are widely scattered, while at other times they are aggregated into tumors which may show simple dilated lymph vessels or definite cavernous spaces, or both conditions combined. Further information on the minute structure of these lymphangiomata is needed.

The fact that the chylous effusions may alternate and even concur in the same individual points to closely related or identical causation. Thus cases are on record of chylous ascites concurring with chylothorax, and of chylous effusions from the skin alternating or

concurring with chyluria and chylous ascites. The vesicles on the skin from which chyle or lymph are discharged are recognized to be but the boundaries of more deeply seated lymphangiomata; where there are several of these vesicles, a few of them discharge at a time, then perhaps become sealed up, and after an intermission which may be short or long, others in turn open the way for escape for the chyle. May not this external lymphorrhœa have a counterpart on internal mucous surfaces?

Further evidence of the close alliance of these effusions may be found in their common etiological relationship to filariasis, or the presence of filariæ in the blood. All of them can be produced by filariæ, whether very frequently as in the case of chyluria, or infrequently as in the case of chylous ascites. Of the existence of lymph varices and lymphangiomata situated internally, the chief, if not only, clinical evidence is afforded by chylous or lymphous effusions. And as escape of lymph is not a necessary consequence of their existence, they may probably be present for a long time, or even always, quite unbeknown to their possessors. In support of this is the well-known fact that the blood may be infested with filariæ without inconvenience or impairment of health.

We will now consider the second group of consequences which may follow obstruction of the thoracic duct or its venous outlet. Instead of the increased pressure on the distal side of the obstruction leading to dilatation of lymph channels and lymph varix, the thoracic duct itself gives way under the strain and perforates. It will be at once seen that this is a much more grave condition than the formation of lymph varices, for while in the latter case collateral circulation is established and some chyle reaches the blood stream, in the present case the line of least resistance is of necessity towards the perforation, all or most of the chyle escapes, and the body is deprived of an important part of its nourishment. The effusion further collects much more rapidly and may set up severe pressure symptoms. The effusions which are caused by rupture of the thoracic duct are limited to the neighborhood of that vessel and therefore must take place into either the peritoneal or the pleural cavities. A case of chylous ascites recorded by Whitla (*British Medical Journal*, 1885, I., p. 1,089) illustrates this condition to perfection, both in its clinical and pathological aspects.

The patient was a boy, aged 13, whose father had died of phthisis. His illness began somewhat suddenly with rigor and pain in the left side. A fortnight later swelling of the abdomen was marked, though there was no œdema elsewhere and there were also anorexia and thirst, but no pain. The ascites went on increasing and doubtful signs of tubercle appeared in the chest. The first para-

centesis was performed and the fluid was found to so resemble milk as to be mistaken for it, unless closely examined. The relief was only temporary, the abdomen rapidly refilled and the paracentesis was repeated with the same results. After this, tappings succeeded each other at short intervals. The fluid was investigated by Professor Hay and declared to be true chyle; the total quantity of fluid thus removed was enormous, amounting to fifteen gallons (English) in sixty-eight days, or an average of thirty-five fluidounces per diem.

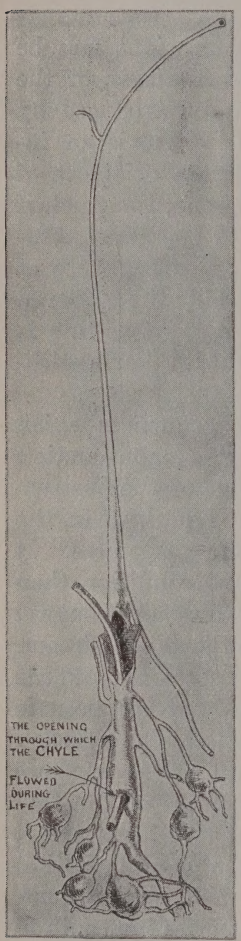

FIG. 43.—The Thoracic Duct in Whitla's Case.

The patient during this time progressively lost flesh and strength, the signs of tuberculosis became more apparent and more general, those of meningitis marking the final stage. The post-mortem examination showed the peritoneum to be thickly studded with miliary tubercles, and the condition was especially marked over the mesentery. The lungs were affected with miliary tuberculosis and the left pleural space was obliterated.

A special dissection of the thoracic ducts was made by Professor Redfern and his description will be closely followed.

The *lower third* of the duct was considerably dilated and the walls were of the normal thickness. Upon its internal surface were minute elevations, which microscopical examination showed to be miliary tubercles. At the lower end of this segment, and at the commencement of the duct, was a perforation, with rounded margins, which admitted a No. 2 catheter (Fig. 43).

The *middle third* in its lowest part contained a plug of fibrin, its lumen was equal to the diameter of a fine knitting needle, and minute tubercles were found on the internal surface. The upper part of this segment was quite impervious, and when sections were examined under the microscope by Dr. Maguire the remains of a lumen were seen, surrounded by thickened fibrous walls in which minute tubercles could be made out.

The *upper third* was more or less impervious except at the extreme upper end which was quite normal.

The cause of the chylous effusion in this case was clearly obliteration of the thoracic duct followed by increased pressure, and dilatation and rupture on the distal side.

It is quite possible that some local weakening of the wall by

tubercular deposit helped to determine rupture of the duct as a consequence, rather than peripheral lymph varix.

Another case in which the consequences were the same though the cause was different is recorded by Cayley (Transactions of the Pathological Society, London, XVII., p. 163). A lad, aged nineteen, was admitted for abdominal pain and tenderness of recent and acute onset; the following day vomiting set in, the patient became collapsed and rapidly sank. At the autopsy the abdominal cavity was seen to contain a yellow fluid. In front of the spine the peritoneum was pushed forward by a milky effusion, which extended downwards behind the serous membrane as far as the brim of the pelvis. The greater part of the thoracic duct was dilated to the size of a little finger and contained a milky fluid resembling that behind the peritoneum. The receptaculum chyli was much dilated and on its anterior surface there was a definite rupture. Near its junction with the subclavian and jugular veins the thoracic duct became narrowed and its coats were thickened, and this part of its lumen was completely occluded by a firm yellow clot. At the mouth of the duct was a granular vegetation almost completely blocking the outlet. The obstruction of the outlet by the vegetation was probably of some standing and had caused the dilatation on the distal side, while the clot was of recent and sudden formation and was responsible for the rupture. It is difficult to explain why such severe peritoneal trouble should have so rapidly followed the rupture, for, judging from other cases of chylous ascites, the peritoneum tolerates well contact with chyle.

The cases of thoracic-duct obstruction followed by rupture of the main trunk on the distal side of the obstacle are very few, and though theoretically possible no case of chylothorax has been recorded as due to this cause.

Causes of obstruction of the thoracic duct may be classified as follows:

(1) Pressure from without.
(2) Inflammation, infiltration, or thickening of the walls.
(3) Blocking of the lumen by
    (*a*) Thrombi;
    (*b*) Morbid growth;
    (*c*) Parasites and foreign bodies.
(4) Filaria sanguinis hominis nocturna.
(5) Blocking of the venous outlet.
(6) Congenital absence or stenosis.

In pressure from without, tumors would be the agents and in a few instances have been observed. In the case of universal lymphangiectasis with lymphorrhœa quoted by Unna (p. 654), the cause of the stenosis was a glandular tumor.

In another case pressure of carcinomatous glands on the lower part

of the thoracic duct contributed to the production of chylous ascites (Weiss, *Centralblatt für innere Medicin*, 1894), and enlarged bronchial glands were the cause in a boy in whom chylous ascites followed croupous pneumonia (Van Swieten, "Comment.," IV., p. 189). Under heading (2) of the classified causes would be included cicatricial contraction following injury or inflammation, and thickening from tuberculosis. The latter was the cause of occlusion in Whitla's case already quoted.

Thrombosis of the duct may follow injury or result from inflammation or other disease of the wall as in the case of veins. On three occasions, Stilling observed local deposit of tubercles in the duct with associated thrombi (*Archiv für pathologische Anatomie*, Virchow, 1882, Vol. LXXXVIII., p. 111). Thrombosis is the natural result of the stasis produced by occlusion of the left innominate or the terminal portions of the left jugular and subclavian veins, and is well illustrated by the cases of Martin and Ormerod.

The obstruction of the thoracic duct by the parent worm filaria sanguinis hominis nocturna is placed under a separate heading, partly because of its great importance as a cause and partly because it is not quite clear how far it effects obstruction by blocking the lumen and how far by setting up inflammatory changes in and around the wall of the duct.

Of chylous effusions all may be associated with the presence of embryo filariæ in the blood and lymph, it may be almost invariably as in the case of chyluria, it may be frequently as in the case of chylous skin discharges, or infrequently as in chylous ascites. Liability to lymphatic diseases such as chyluria is met with especially in those tropical and subtropical districts where filarial infestation is rife, the two conditions are in short co-endemic. Taking chyluria as an example of the rest, we have already seen that the presence of chyle in the urine forces us to the conclusion that the thoracic duct is obstructed, that the chyle, unable to drain into the venous system by the usual channels, regurgitates along the visceral lymphatics and reaches the blood by a new and devious route; that the raised intralymphatic pressure and regurgitation produce a lymph varix, and that leakage from this produces chylous effusion. Is there anything then to connect causally the filariæ with thoracic-duct obstruction? The presence of innumerable embryos of filaria nocturna in the urine and lymph would point to a parent filaria being situated in the lymphatic system somewhere higher up the lymph stream. Two post-mortems on chyluria cases, those of Drs. Mackenzie and Curnow, have shown the thoracic duct to be obstructed, and although no traces of the parent worms were found at the obstructions, there were

circumstances in each of the cases which might properly explain their absence; and further, although absent in these, the parent worms have been discovered in other lymphatic varices.

To meet the objection that the filarial embryos might pass into the lymphatics from the blood-vessels, Dr. Manson has pointed out that the structure of the embryos ill adapts them to migrate through vessel walls, and further he has shown that sometimes ova which have not yet developed into free embryos and which are incapable of any active movements are found in the lymphatic vessels. We may conclude then that the parent filaria nocturna can become located in the thoracic duct, that when there it sets up obstruction, and that varix with or without effusion follows. The fascinating subject of filarial parasites and their dealings with man does not fall within the scope of this contribution and is fully treated elsewhere in this work. It is verily a fairy tale of science, which various workers and especially Manson have step by step unfolded to us.

Congenital stenosis of the thoracic duct is mentioned by one or more authorities as a cause of lymph stasis, but a careful search has not revealed any undoubted example of this condition.

Chylous Effusion and Heart Disease.—There is a body of evidence connecting some chylous effusions with valvular disease of the heart. Under normal conditions of the circulation the outflow from the thoracic duct is much aided not only by the low pressure subsisting in the large veins, but also, on a well-known mechanical principle, by the flow of venous blood heartwards past the duct outlet. It must follow then in cases of morbus cordis, where there is much back pressure and the large veins are over-distended, that the outflow of lymph from the thoracic duct is impeded and the lymphatic pressure raised; and there is no doubt that the ills resulting from dilated heart are due not only to engorgement of the veins but largely if not equally to obstructed circulation in the lymphatics.

It is not unreasonable to suppose then that in rare instances the back pressure in the thoracic duct may from this cause be of such a degree as to produce thrombosis and, following this, lymph varix and effusion.

Oppolzer (*Allgemeine Wiener medizinische Zeitung*, 1861, p. 149) as far back as 1861 described a post-mortem on a case of morbus cordis, complicated with chylous ascites, in which the termination of the thoracic duct was blocked by a pale red thrombus and the duct below the obstruction was thickened and dilated. Incompetence of the mitral and tricuspid valves was also demonstrated.

Rokitansky ("Lehrbuch der pathologischen Anatomie," 3d edition, 1865, Vol. II., p. 388) gives a very full account of chylous ascites and

chylothorax complicating dilated heart from mitral disease in a woman aged sixty-two. At the autopsy, there were chylous effusions in both pleural cavities with dilatation of the subpleural lymphatics, and there was also extensive chylous effusion in the abdomen. The efferent lymphatics from the small intestine were for the most part dilated, varicose, and filled with a creamy substance. The receptaculum chyli was dilated and its walls thickened, the thoracic duct was in some places dilated and filled with a creamy material similar to that found in the intestinal lymphatics, and in other places was quite obliterated by fibrous contractions.

The following case will be of interest as illustrating the same condition of lymph varix associated with a congenital heart lesion. It is recorded by Cholmeley (Transactions of the Clinical Society, London, 1869, II., p. 116).

A child had the usual signs and symptoms of a congenital heart lesion. At the age of six, when these signs were still present, an elephantiasic swelling of the right leg and ankle appeared and, spreading very slowly, had after a further lapse of about three years involved the whole of the right lower extremity. From papules which formed in the neighborhood of the ankle a milky alkaline fluid exuded.

Another though different example is worthy of mention. Petters (*Prager Vierteljahrschrift*, Vol. LXXII., 1861) relates the case of a woman with marked mitral stenosis, who was found to have a tumor, the size of a small apple, resembling a hernia and occupying the left inguinal ring. Post mortem, the tumor was found to be a lymphangioma which was so distended with lymph that, on pricking it, a jet of fluid spurted forth. Numerous other dilatations of lymph tracts were found. These examples serve to illustrate the interesting association between morbus cordis and lymph stasis.*

Symptoms.

These may be quite absent. Internal lymph varices and lymphangiomata manifest themselves clinically only when leakage occurs by transudation or fistulæ, and then the symptoms are those caused by the particular variety of effusion set up, and will be considered under their respective headings.

In the *British Medical Journal*, 1890, I., p. 421, is a report of the case of a man who fell ten feet from a scaffold on to some stones. One specimen of typically chylous urine was passed two or three hours after the accident, but the next day the urine was normal. This rare if not isolated instance of traumatic chyluria can be explained on the assumption that the patient already had a lymph varix and that the accident caused a temporary escape of chyle.

External varices and lymphangiomata resulting from thoracic-

* Vide a paper by Busey (American Journal of the Medical Sciences, xc., p. 373).

duct obstruction, besides being visible and exuding chyle, are liable to inflammation from injury, irritation, or pressure.

Treatment.

The *prognosis* and *treatment* of obstruction of the thoracic duct will necessarily vary according to the nature of the clinical manifestations.

Chylous Ascites.

Etiology and Pathology.

The causes of chylous ascites may be arranged as follows:

(1) Obstruction of the thoracic duct or its venous outlet followed by rupture or by varix and leakage.

(2) Back pressure from the large veins.

(3) Traumatism followed by rupture or thrombosis of the thoracic duct.

(4) Affections of the efferent lymph vessels from the small intestine or of their glands, acting in one or more of the following ways:

(*a*) Pressure on the vessels by means of multiple nodules, *e.g.*, of carcinoma or tubercle, or by a thickening of the mesentery due to chronic peritonitis;

(*b*) Blocking of the lumen of the vessels by morbid growths;

(*c*) Disease (*e.g.*, carcinoma) of all or most of the glands of the mesentery, causing obstruction and back pressure in the lacteal radicles;

(*d*) Disease of the walls of the lacteal vessels (*e.g.*, infiltration with tubercle or malignant growth) producing degeneration and thinning and escape of chyle either by transudation or rupture.

Any extensive obstruction of the efferent intestinal lymph vessels will cause a rise of pressure in the chyle vessels and radicles and a tendency to leakage. Of the above groups, the first two have already been discussed.

Traumatism of the thoracic duct as a cause of ascites is illustrated by a case reported by Quincke (*Archiv für klinische Medicin*, XVI., p. 121).

A man with an alcoholic history was, while lying on his side, run over by a cart. A week after the accident, the patient came under treatment for what turned out to be right chylopneumothorax. The chest was aspirated and 1,800 c.c. of blood-stained and typically chylous fluid were obtained. Temporary relief followed, but extensive subcutaneous chylous œdema, commencing at the point of puncture, appeared and a little later the signs of fluid in the pleura returned. A second paracentesis was performed and over three litres of milky fluid were obtained, but notwithstanding temporary relief the

patient sank. Post mortem the thoracic duct was found filled with coagulated blood throughout. There was an effusion of chyle into the peritoneal cavity. The right pleura contained 7,000 c.c. of chylous effusion and there was a simple effusion in the left chest. The lungs were healthy.

As showing the relation between ascites and morbid conditions of the intestinal lymph vessels another case recorded by Quincke may be related.

The patient was a woman, aged 30, who gave a history of hard œdematous swellings of the right leg and forearm since the age of 15. On admission there were hard œdema of the legs, arms, and abdominal wall and considerable ascites. The patient was weak and ill. The abdomen was tapped and ten litres of chylous fluid were obtained. The fluid reaccumulated rapidly and six further tappings succeeded one another at short intervals. The patient's condition did not improve and she died from exhaustion. At the autopsy the thoracic duct was found to be normal. There was much chylous effusion in the abdomen. The chyle vessels of the small intestine were dilated and injected and between them were patchy extravasations of chyle; the vessels were injected as far as the attachment of the intestine to the mesentery, but not in the latter. Here the stasis of chyle with its consequent escape was thought to be due to partial occlusion of the vessels from pressure of a thickened mesentery, the result of chronic peritonitis. But it must be confessed that this explanation leaves out of account the long standing hard œdema of the limbs and abdominal wall and is thus hardly comprehensive enough to be satisfying.

F. J. Smith (Transactions of the Pathological Society, London, XLII., p. 100) assigns multiple occlusion of intestinal lymph vessels from old chronic peritonitis as the cause of chylous ascites in a patient who died of carcinoma.

Strauss gives a very full account of a case in which the cause was cancer of the peritoneum (*Archives de Physiologie*, VII., 370).

The patient was admitted into the hospital complaining of progressive loss of flesh and strength and of dyspeptic symptoms which had begun four months previously. On admission there was much fluid in the abdomen, and much distress resulted therefrom. Paracentesis was performed and six litres of chylous fluid were withdrawn. While the abdomen was lax, several hard nodular tumors could be detected. The abdomen rapidly refilled, rendering a second tapping necessary, which had to be followed by a third five days later. Death occurred before the fluid had time to reaccumulate. Between the second and third tappings the patient was placed on a special diet of butter and milk, with the result that the latter effusion in comparison with the former contained three times as much fat and also constituents which pointed to the presence in it of butter—"la butyrine était trois fois plus forte dans la liquide de la dernière ponction."

At the autopsy the primary lesion was scirrhus of the pylorus and the secondary carcinosis of the peritoneum. The mesentery and retroperitoneal glands were hard and transformed by cancer growth. The mesentery was thick and retracted, and along the line of its attachment to the small intestine was a series of cancer nodules which doubtless obstructed the flow of chyle. On the anterior aspect of the mesentery were discovered two small fistulous openings out of which chyle could be pressed and which without doubt existed during life. Beneath the serous surface of the small intestine, along its whole extent, were numerous white patches of extravasated chyle, and somewhat similar white patches were to be seen beneath the mucous surface; this condition was seen only in the small intestine.

In this case the chyle escaped in greater part through actual fistulæ and in smaller part by transudation through degenerate walls; the obstruction was caused partly by the mesenteric glands and partly by pressure on, and obliteration of, the efferent chyle vessels. Incidentally such a case shows that secretion from the intestine into the central lacteals of the villi can take place under a very considerable pressure in the latter.

Symptoms.

The symptoms are firstly those of ordinary ascites. When there is no actual perforation and the obstruction is not severe, the fluid will be moderate in amount and will accumulate slowly. Under such circumstances there would be little specially to attract the attention. Where an aperture exists, and especially if it exist in a large trunk like the thoracic duct with severe obstruction on the cardiac side of it, the amount of fluid is great and accumulates rapidly. In such a case anæmia, loss of flesh and strength, and other symptoms pointing to a gravely impaired nutrition, and resulting from the loss to the body of an important nutritive fluid, appear in addition to those of ascites. Whitla's case quoted above illustrates this more severe form. In addition there will sometimes be the symptoms of the primary disease.

Diagnosis.

Unless there be a concurrent chylous effusion elsewhere, *e.g.*, from the skin or in the urine, no diagnosis between ordinary and chylous ascites is possible until the fluid has been withdrawn. The symptoms arising from the causal disease might aid, but would probably more often embarrass the investigator.

True chylous effusion has to be distinguished from a pseudo-chylous or chyliform ascites in which the fat has been formed in the peritoneal cavity by the fatty degeneration of cellular elements, and

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several cases placed under the former category in reality belong (as Bargebuhr, Busey, and others have shown) to the latter.

A careful examination of the fluid, and the amount and the peculiar molecular form of the fats, aided if necessary by the results of experimental feeding, would enable a diagnosis to be made.

Prognosis.

This is of necessity grave, on account of the condition itself and often also because of its cause.

Escape of chyle through a definite rupture, unless perhaps the latter be traumatic, is more grave than escape by multiple leakages, and the larger and more central the trunk the more serious the prospect. The most hopeless case then of ascites quâ ascites would seem to be one where the thoracic duct is obstructed above and ruptured below, for not only is the escape of chyle very rapid but, owing to the fact that the direction of back pressure is toward the fistula, no establishment of a collateral circulation is possible. The formation of an effective anastomotic circulation is a favorable circumstance, for the raised intralymphatic pressure is released and the chyle is saved to the body. If the escape of the chyle be peripheral, the smaller the area obstructed the less serious will be the prognosis.

Treatment.

Of this little can be said. Rest is frequently a necessity from the patient's condition, and apart from this it tends to diminish the escape of chyle, and possibly in rare cases to favor healing of a fistula if such exists.

Tapping should not be resorted to unless pressure symptoms render it necessary. It would favor rather than not the escape of chyle from the vessels. Inunction of mercurial ointment has been suggested by Lancereaux. For the rest, the treatment is palliative and directed to the maintenance of the patient's strength.

In filarial cases, which form but a small fraction of those recorded, parasiticides have been advocated, but they would be useless if not, indeed, harmful.

Chylous vomiting and chylous diarrhœa sometimes complicate cases of chylous ascites, and in the light of what has been set forth above, their mode of production is easily intelligible. In a case recorded by Pelletier (*Journal de Médecine, de Chirurgie et de Pharmacie*, Vol. LXIII., p. 496) chylous ascites, chylothorax, chylous vomiting, and chylous diarrhœa concurred in the same patient.

Chylothorax.

Only a few undoubted cases of this very rare condition have been recorded. The modes of its production resemble those of chylous ascites, excluding those forms of the latter whose causes lie within the abdomen. Thus it may be produced by traumatism, by blocking of the thoracic duct or the veins into which the latter empties, or by back pressure from the large veins.

There are examples of each of these modes of formation. In the case recorded by Quincke the duct was ruptured by the passing of a wagon over the thorax. Martin and Ormerod each found thrombosis of the veins on the left side of the neck, and in another instance (Rokitansky) increase of venous pressure from valvular disease of the heart was the cause. The symptoms and signs show no differences from those of an ordinary pleural effusion, and unless there were concurrent chylous effusion elsewhere, no diagnosis would be possible until the fluid was drawn off. In one or two instances, where the chylothorax was secondary to chylous ascites, it has been suggested that the chyle traversed the lymph vessels of the diaphragm, but it is difficult to see why the fluid should take so fanciful a journey. The treatment in no way differs from that of hydrothorax.

Chylocele.

Chylocele or effusion of chyle into the cavity of the tunica vaginalis is most frequently, if not invariably, an evidence of filarial disease, with other forms of which, lymph scrotum, chyluria, etc., it is often associated. In its character, chylocele resembles an ordinary hydrocele and its nature is often apparent only when the fluid is withdrawn. The fluid has the characteristics of chylous effusions; it possesses the power of spontaneous coagulation, contains albumin, finely divided fat, and a large number of the embryos of filaria nocturna. It is produced by the rupture of varicose lymphatics and has been cured (Martin quoted by Busey) by dissecting back the varix and then applying a strong ligature.

Chyluria.

This is a milky condition of urine due to the presence of chyle. It is for the most part a tropical disease, and, in the majority of cases, is associated with the presence of filariæ in the blood.

The Urine.

In a typical case, if such exists, the urine when passed has the consistence and color of milk, its smell and taste are sweetish and in no way urinous; its specific gravity is often below the normal but seldom above it, varying from 1.010 to 1.025. As to quantity, in many cases no precise observations are recorded, but sometimes polyuria, *e.g.*, one hundred and twenty ounces, has been noted. The reaction is slightly acid, neutral, or slightly alkaline. The urine is markedly albuminous and on boiling a coagulum forms which entangles fat as it falls. If the chylous urine be slightly acidulated and then boiled and finally filtered, the filtrate resembles urine except that it is still slightly opalescent.

In a brief period after it is passed, generally a few minutes but sometimes extending to a few hours, the milky urine sets solid and forms a tremulous coagulum resembling junket, and having the shape of the containing vessel. Sooner or later the coagulum contracts and breaks up, and the contents of the vessel are again for the most part fluid. A scum of creamy consistence rises to the surface, a reddish deposit falls to the bottom, and there is an intermediate stratum, often of a pinkish color, in which floats the contracted clot. The creamy top stratum is soluble in ether and under the microscope shows fat, in the form partly of globules and partly of small granules, similar to the molecular fat found in chyle. The deposit consists of fat globules and granules, lymph cells, red blood discs, and most often, except in the few non-parasitic cases, filarial embryos. Casts are absent with rare exceptions. The filariæ may be found by teasing bits of the contracted clot, but according to Manson the best way of finding them is to break up a newly formed coagulum, allow the urine to stand, and then examine the sediment. If the milky urine be shaken with ether it is clarified, and if the fluid below the top stratum be pipetted off it is found to contain but little fat, but still to be rich in albumin.

A large proportion of chylous urines contain blood, and frequently the fresh urine has a pinkish hue due to this cause, but the amount is small. In some cases recorded, attacks of hæmaturia have occurred between those of chyluria, but were probably due to a concurrent and not to the same morbid condition. Not all chylous urines are equally milky; they vary much, as chyle does, in the amount of fat they contain. The specimens which are very poor in fat often resemble pale amber in appearance and are styled lymphous (Roberts); in other respects, such as in regard to coagulability, they resemble the more milky specimens.

It is not uncommon for coagulation to take place within the bladder, in which event clots are passed, and their passage may be preceded and accompanied by pain; there may even be a temporary retention of urine.

The condition is fitful both in its coming and its going. The urine will be chylous for days, weeks, or months and then become normal for like periods. When once a patient has suffered from chyluria, recurrences are the rule. Further than this, the amount of chyle often varies with the time of day; the morning urine may be rich in chyle and the urines during the day more or less free, or the opposite may prevail. The influence of the time of day on the amount of chyle in the urine varies with different patients, but seems to be roughly constant in the same patient. Thus it is often recorded that the urine is least milky or quite clear in the morning after the night's rest; on the other hand, in a case reported by Beale, the patient passed chylous urine only on rising in the morning, and, in a non-filarial case reported by Siegmund, the chylous urine was usually passed between 5 and 6 A.M. Exercise seems often to increase the amount of chyle, as also does the recent ingestion of food, whereas starvation reduces the quantity; but these are very far from being unvarying sequences.

Symptoms and Duration.

There may be no symptoms at all, and but for the knowledge of the chyluria the patient would be judged to be in good health. A case is on record of a woman who died at the age of eighty after having had chyluria intermittently for fifty years. On the other hand there are commonly weariness, weakness, and debility with pains and dragging in the loins, extending sometimes to the thighs, groins, and testicles. There may be tenderness on pressure over the loins or abdomen; this was noticeable in the Brazilian case recorded by Havelburg.

When chyluria persists for long or with severity, marked anæmia and debility result. Where death had occurred, it has usually been from some intercurrent disorder. In the parasitic cases, however, there is the possibility of a fatal issue from the disease or death of the parent filaria.

Treatment.

Of treatment which can have any claim to be reliable in its results there is none. The advisability of abstention from physical exercise and of rest in the recumbent position would seem to be supported by

both pathology and experience, but where the symptoms are slight it would be impracticable of application.

It is recommended that the diet should contain very little fat with a view of diminishing the amount of fat in the urine. There is no doubt that the amount of fat in chyle promptly varies with the amount of fat in the food, but it is not clear what advantage would accrue from freeing the urine of fat as long as the albuminous constituents of lymph escape in undiminished quantity; it is simply the difference between a clear and a milky clot.

Gallic acid in half-drachm doses has had a partial success in some hands, and Mackenzie has recorded good results from benzoate of soda, up to two drachms thrice daily.

Among other remedies turpentine, iron, and potassic iodide have been tried without results.

Injections into the bladder have had their advocates, and in one case recorded by Dickinson injections of iron seemed to do good.

Of specific anthelmintics there are none and those that have been suggested are held by Manson to be not only inefficient in purpose but also undesirable in aim, on the ground that a dead filaria is more likely to be harmful to its host than a living one.

Two cases published by Laurie (*Lancet*, 1891, I., 364) held out hopes that thymol in two-grain doses was an efficient drug in chyluria, but subsequent experience has sent this remedy the way of its predecessors.

LYMPHANGIOMATA.

Lymphangiomata may be defined as tumors composed of dilated lymph vessels. On section they are seen to consist of smaller and larger dilated spaces which are lined with endothelium and separated by a varying amount of interstitial tissue.

They may be either congenital or acquired.

According to the size, shape, and arrangement of the spaces they are divided by Wegner and others into three forms:

1. Lymphangioma simplex;
2. Lymphangioma cavernosum;
3. Lymphangioma cysticum.

Frequently two of the forms with transitions between them are represented in individual tumors, and there is a degree of resemblance in the structures of the three forms which probably represent different degrees of the same development.

In *lymphangioma simplex* the spaces are small and consist of a number of dilated and anastomosing lymph vessels which, if distended, are visible to the naked eye.

In *lymphangioma cavernosum* the spaces are larger, varying from the size of a pin's head to that of a cherry, and the degree of varix is more marked; the whole tumor has the appearance of a sponge, and consists of numerous anastomosing spaces separated by a trabecular framework of connective tissue.

In both these forms the spaces are lined with endothelium and communicate with the lymphatics of the neighboring healthy tissues, so that under pressure they may be partly emptied of their contents. The latter consist of chyle, lymph, or of lymph thrombi. The stroma is made up of fibrous tissue, sometimes fully developed and sometimes showing many spindle cells, a proportion of elastic fibres, and some infiltration of round cells which are scattered or collected into definite lymphoid deposits. Less frequently we find fatty tissue entering into the composition of the growth. The thickness of the trabeculæ varies much. The stasis of lymph always produces some hypertrophy of the vessel walls and of the interstitial tissue; sometimes it is slight, while at other times, owing probably to recurring attacks of inflammation, it is very great, extending far and wide and so producing those giant growths which are aptly denoted by the term elephantiasis.

Lymphangioma cysticum has the appearance of a number of cysts of varying sizes bunched together so as to form a tumor. The contents of the cysts are lymphous, and though these cysts have lost all external resemblance to lymphatic vessels, they are the result of their extreme dilatation, and are in the majority of instances developed from cavernous spaces. They are lined with endothelium, but unlike the spaces of the preceding form, the communications between them and the surrounding lymphatics are very limited or even wholly absent, and therefore the tumor cannot be diminished by pressure.

The larger cysts are produced by the confluence of two or more of the smaller ones, and such confluence may be so extensive that the tumor may become a large unilocular cyst.

Modes of Formation.

Of these there are three:

1. Dilatations of pre-existing lymphatics, by occlusion of efferent lymph trunks, with increase of connective tissue around and between the dilated vessels—ectasy with hyperplasia.

Macroglossia illustrates this mode of formation.

2. By proliferation of the endothelial cells of the original lymph capillaries. Small buds or outgrowths from the endothelium appear on the sides of the vessels; these increase and form columns of cells

which fuse with similar columns growing from other lymph vessels. Clefts then appear in these cell masses, and the latter gradually become canalized by new lymph channels, which communicate with one another and the older vessels. At the same time the pressure of the new cell growth causes the diminution and even the disappearance of the original connective tissue between the meshes of the lymphatic network.

As an example of this mode of formation Wegner gives the following case:

A man, aged 49, complained of a small tumor in his forehead which was slowly growing. On examination, the growth was seen to be the size of a medium-sized tomato and pseudo-fluctuating; on removal, it discharged a glairy fluid, and on microscopical examination it was found to be a lymphangioma cavernosum situated in the corium and subcutaneous tissue; the earlier changes were to be seen near the surface of the tumor, and the rich development of new vessels and proliferated cell growth in the deeper part. The isolated cutaneous lymphangiomata are formed on this type.

3. A third and heteroplastic mode of formation is described (Virchow and Winiwarter). Granulation tissue springs up in the connective-tissue matrix, and by a secondary transformation new lymph spaces are formed.

Of the above, the first is the most frequent and, from its clinical results, the most important mode of formation.

That lymphangiomata can be acquired as the result of obstruction of the thoracic duct and that they can be experimentally produced by a prolonged maintenance of raised intralymphatic pressure has already been amply shown.

There are equal grounds for expecting that obstruction of the lymph channels of local areas would be followed by like results, and we do find that occlusions of the lymphatics of the leg or arm, for instance, whether they be produced by the compression of a growth or by means of filarial ova or in other ways, are followed by lymph stasis and varix on the distal side of the obstruction. The result of occlusion may anatomically be either a diffused lymph varix with hyperplasia, such as is seen in elephantiasis and the elephantiasic condition, or a circumscribed and even encapsuled tumor. In the acquired form the causes of the obstruction of the lymph trunks, viz., compression from without, thickening and contraction of the walls, and blocking of the lumen by thrombi or filariæ have frequently been demonstrated.

The same cannot be said of the corresponding variety of congenital lymphangiomata, and their causes are matters for the most part

of inference rather than of demonstration. Their resemblance to the acquired form and the fact that their favorite situations, *e.g.*, the neck, tongue, and groin, are often those where kinking and thrombotic occlusion could easily occur, are facts which support this view of their origin.

The chief factor then in the production of this class of lymphangiomata is obstruction of the efferent lymph trunks; the result may be either a diffuse infiltration or a circumscribed tumor, the situation may be either internal or external, and there may or may not be an accompanying lymphorrhœa. It should here be mentioned that so high an authority as Unna dissents emphatically from the view that lymph stasis following obstruction can alone produce ectasia; he holds that in every case there must be active proliferation. This author's views are expressed with great definiteness in the following quotation: "A simple obstacle on a lymph passage cannot produce either œdema or lymphangiectasis or lymphangioma or fibromatosis." Granting that there must be an active proliferation from the endothelium in this as in the second mode of formation, it will still remain that lymph stasis caused by occlusion will be a necessary antecedent condition in the former but not in the latter variety.

A few illustrative examples of these tumors will now be considered. A large proportion of them are congenital.

Macroglossia.—This is an important representative of the mixed simple and cavernous lymphangioma. If a section of such a tongue be examined it is seen to be permeated in all parts by cavities and canals of various sizes and shapes which communicate with one another and have the characters of lymph vessels.

Between the dilated lymph spaces are a varying amount of connective tissue rich in spindle cells, here and there aggregations of lymphoid cells, and the muscular fibres proper to the tongue. The character of the intervening tissue varies, a large proportion of spindle cells representing activity of growth, and a more matured fibrous tissue the reverse. It is chiefly on the amount of this connective tissue that the size of the organ depends; where it is considerable the muscular fibres undergo considerable attenuation from compression.

In its production the growth illustrates both the first and second modes of formation described above; primarily it results from occlusion, lymph stasis, varicose dilatation of pre-existing lymph vessels and hyperplasia; and secondarily from an active endothelial proliferation with formation of new lymph vessels. In the more moderate degrees of enlargement the tongue presents such appearances as are above described: it is riddled with clefts and cavernous spaces which are separated by a moderate amount of interstitial tissue; in some

instances the loculi of the tumor may be near the surface and appear as papules or even vesicles on the dorsum, from which lymph may from time to time be discharged. But an enlarged tongue is constantly exposed to the irritation of slight and unnoticed injuries, and owing to this and the lymph stasis the organ is constantly the seat of slight and recurring attacks of inflammation. The resulting hypertrophy contributes largely to the later and progressive increase of the organ and it follows that in many, if not all, instances macroglossia when it comes under observation is the result not of lymphangioma alone, but of lymphangioma plus hypertrophy, the latter resulting from recurring though perhaps unnoticed attacks of inflammation predisposed to by the former. Where the giant growth of the tongue is due to these combined causes we get a condition bearing a close resemblance to elephantiasis in which the same two causes are at work. Although a congenital condition, the enlargement is at first often slight and may not be noticed for the first few years of life, or in rare instances till a still later period. When it is noticed it means that hypertrophy of the organ is slowly progressing. It follows then that macroglossia may show degrees of severity varying from a hardly noticeable inconvenience to an elephantiasic enlargement which displaces the teeth and impairs articulation, and which, in one instance mentioned by Morgan, protruded six and a half inches beyond the upper lip and measured ten inches in circumference at its base. The connection between macroglossia and the lymphangiomata is well shown in a case recorded by Winiwarter in which a congenital cystic lymphangioma of the neck was associated with macroglossia, the lymph spaces of the two tumors communicating with each other.

Macrocheilia.—This is a condition, similar both in structure and origin to macroglossia, affecting the lips, the upper more often than the lower. The two conditions are sometimes associated.

The following case (zur Nieden, Virchow's *Archiv*, 1882, p. 350) is one of lymphangioma simplex cavernosum and its mode of formation resembles that of macroglossia.

A girl at the age of nine noticed for the first time that she was losing a milky fluid from the external genitals and on close examination she found that it escaped from vesicles situated on the large labia; for eleven years she sought no advice, and during this interval the discharge was uninterrupted, though it could be almost made to disappear by complete rest. When, at the age of twenty, she first came under observation the large labia were found to be moderately enlarged and beset with vesicles which could be emptied by pressure, and became much distended after exercise. Her symptoms were much relieved by rest and she left the hospital, but later had to be

readmitted for a return of her trouble. For purposes of diagnosis a small fragment, which included the whole thickness of the cutis, was removed from one of the labia. This slight operation was followed by a considerable lymphorrhœa from the wound which lasted several days, and amounted in the course of one night to 160 c.c. On examination the fluid was found to have all the characters of chyle. Sections of the removed fragment showed the changes to be chiefly situated in the cutis. Here were seen a number of anastomosing spaces of various sizes and shapes; beneath the epithelium they were small and cleft-like, and in the deeper parts larger and of a cavernous character. Canals entered some of the cavities from below, and both they and the spaces were lined with endothelium. The tumor was treated with the cautery and a complete cure resulted, though whether it was permanent is not stated. This growth then was situated in the cutis; nevertheless it was not circumscribed, as a large number of the cutaneous lymphangiomata are, but was probably part of a more extensive ectasia. We are justified in the latter conclusion from the fact that true chyle escaped from the surface of the growth and from the wound produced by the diagnostic operation: and the presence of this fluid in the lymphatics of the skin can only be explained by supposing the existence of an obstacle to its flow into the thoracic duct and by its journeying toward the blood stream along devious and unusual routes. The mode of formation of this lymphangioma was, as in the case of macroglossia, occlusion followed by stasis and ectasia, and probably later by proliferation and formation of new lymph vessels. In origin it was most likely congenital, but remained unnoticed until the lymphorrhœa attracted the patient's attention.

The lymphangioma of the abdominal wall with lymphorrhœa recorded by Roberts, and already quoted, belongs to the same category as the foregoing. The tumor was probably of the cavernous variety, was situated in both the cutaneous and subcutaneous layers, contained chyle, which was discharged from vesicles on the surface of the skin, formed part of a widely distributed ectasia, and was associated with chyluria. Unlike the last example it was acquired, not congenital, and the cutaneous and subcutaneous tissues in which it was situated showed considerable hypertrophy.

Examples of the same condition and modes of formation are recorded by Petters, Fetzer, Carter, and many others. Petters describes a lymphangioma with milky lymphorrhœa of the left labium majus followed later by elephantiasis of the left thigh. The patient died of intercurrent peritonitis and an extensive lymphangiectasis of the abdominal lymphatics, and especially of those of the pelvis, was found. Carter's case is an example of lymph scrotum associated with chyluria and varicose inguinal glands.

Turning now to the *deeply seated lymphangiomata*, we find that they are of the cavernous and cystic varieties, the two forms often being found together in the same growth. An interesting example of

this variety was found by Weichselbaum (*Archiv für pathologische Anatomie*, 1875, LXIV., p. 145) in the abdomen of a patient who died, aged eighty, of pneumonia. The growth was the size of the palm of the hand, and was situated in the mesentery of the ileum. On cutting into it, the exposed section was seen to be riddled with cavities varying from the size of a pin's head to that of a hazelnut, and freely communicating with one another. The spaces were numerous near the centre and sparse at the periphery; they were imperfectly lined with endothelium and all of them were full of chyle; the interstitial tissue contained much fat. Weichselbaum styles the growth a chylangioma cavernosum. As none of the loculi became thinned there was no chylous effusion into the peritoneum and the possessor of this tumor was quite untroubled by its presence. The tumors found post mortem in the fatal cases of chyluria recorded by Mackenzie and Havelburg, and described above as illustrations of the consequences of thoracic-duct obstruction, are examples of the internal lymphangiomata associated with chylous effusions. In Havelburg's case the tumor consisted of loculi separated by connective tissue and containing chyle; the lower part of the tumor was closely connected with the bladder and one of its loculi communicated with that sac.

The two following cases are instances of internal lymphangiomata extending and presenting through the inguinal canal, outside which they appeared as tumors and were taken for hernias. The first case, reported by Amussat, was in a boy, aged nineteen, who had a tumor in each groin for which he was wearing a truss. The patient became suddenly ill with fever and pain in the right groin and died in a few hours. Post mortem the tumors were found to be masses of dilated lymph vessels continuous through the inguinal canals with similar ectasies of the iliac lymphatics. The second case, recorded by Trélat, was in a boy who also had what was thought to be a double inguinal hernia; the tumors appeared first during exertion. The patient died after a slight operation for fistula, and at the autopsy the tumors were found to be lymphangiomata extending into the abdomen, the two masses converging near the diaphragm. The question might be raised whether these internal lymph varices are rightly termed lymphangiomata. If we adopt the accepted definition of lymphangiomata as tumors composed of dilated lymph vessels, the answer will depend in any given instance on whether the dilated vessels are sufficiently aggregated together to form a tumor. Lymphangiomata of this class, both congenital and acquired, which consist of preexisting lymph channels and result from obstructed lymph flow, are tumors, although they are not neoplasms, whereas the lymphangi-

omata formed according to modes 2 and 3 above mentioned are both tumors and neoplasms.

We will next briefly recur to the *cystic lymphangioma* which was the variety artificially produced by Wegner as explained and illustrated on page 655. It is frequently associated in the same tumor with the cavernous form, but it is best exemplified by certain rare congenital tumors of the neck (cystis colli congenita) and elsewhere. Pieper described such a tumor in the case of an infant. At the age of seven weeks, when the child came under treatment, there was a lobulated swelling the size of a hen's egg situated on the left side of the neck between the mastoid process and the middle line in front. There was a similar swelling on the right side of the neck, only smaller. The tumors were situated in the subcutaneous connective tissue, their size could not be reduced by pressure, and their contents consisted of blood-stained lymph. There was concurrent macroglossia. These neck lymphangiomata are usually superficial and covered only by the skin, but occasionally they have been found deeply situated following the intermuscular connective tissue.

And lastly, there is the group of isolated lymphangiomata of the skin. These formations have been studied mainly by dermatologists, by whom they have been variously named. They are all comprehensively included under the designation *lymphangioma circumscriptum cutis*. They are isolated tumors of a nævus habit, situated in the cutis, for the most part congenital, of the simplex cavernous type, and are formed by endothelial proliferation. Clinically they appear as vesicles like sago grains, and are collected together into a patch or patches. The tumor slowly extends at the periphery by the formation of fresh groups of vesicles.

Symptoms.

These may be absent, the tumor giving rise to no trouble, or at most only causing slight inconvenience. On the other hand it may from its size or position produce pressure symptoms and these even of gravity—for example, dyspnœa, compression of veins, and œdema; it may impair movements, *e.g.*, of the tongue, and cause difficulty in swallowing and articulation. All lymphangiomata, especially those near the surface of the body, are liable to attacks of lymphangitis, with the usual accompaniments of fever, pain, swelling, and tension; these attacks usually subside after a brief space, but not without having caused a permanent increase in the size of the tumor; at times the inflammation instead of clearing up goes on to suppuration, and further trouble results. Lymphorrhœa occurs in a good proportion

of the cases, and in the case of the deep-seated lymphangiomata is the chief objective token of their presence. If moderate it is inconvenient and nothing more, but if it is severe, and especially if in addition it is chylous, weakness and anæmia of varying severity result. In the case of the internal lymphangiomata the symptoms are those of the various chylous effusions, to wit, chylous ascites, chyluria, etc.

Diagnosis.

This is easy in the case of macroglossia and macrocheilia, but in that of other superficial lymphangiomata, in the absence of lymphorrhœa, it is very difficult and often even impossible. Especial mention may be made of lymphangiomata occurring in the inguinal region, where they are liable to be mistaken for inguinal herniæ. The diagnosis of a deep-seated lymphangioma in the absence of a chylous effusion or of an associated lymph varix elsewhere, could not be made.

Prognosis.

The lymphangiomata are benign growths and if completely extirpated do not return. Extirpation in the case of the deep-seated forms is impracticable, except in the case of chylocele, and sometimes in the case of the superficial ones is incomplete, owing to their wide diffusion. Apart from chylous effusions the deep-seated tumors need cause little anxiety. Of the effusions, the prognosis in the case of chylocele is the best and that of chyluria next. Chylous ascites and chylothorax are more grave affections. External lymphorrhœa need cause no anxiety unless it is excessive.

Treatment.

The treatment of the superficial lymphangiomata falls more within the domain of the surgeon than of the physician. The methods adopted are briefly these: Paquelin's cautery, extirpation by the knife, subcutaneous injection of an irritating fluid, such as iodine, and compression. Of these the first two are the more favored. In the case of the deeply seated lymphangiomata no radical treatment is possible except in the case of chylocele, in which the varix has been dissected out and ligatured. Macroglossia is successfully treated by removal of a V-shaped piece from the enlarged organ. For the isolated lymphangiomata of the skin electrolysis has been employed with benefit. For the rest, the treatment is symptomatic.

ELEPHANTIASIS.

In its widest sense elephantiasis is applied to any marked degree of hypertrophy of the skin and areolar tissues of a part; while in its narrower application it denotes a disease, endemic in certain tropical and sub-tropical countries, which is characterized by a recurrence of febrile attacks, each attack being accompanied by inflammatory œdema and progressive hypertrophy of the cutaneous and areolar tissues.

Morbid Anatomy.

The integuments of the affected part, say the leg, are thick, indurated, and often rough and cannot be made to move over the more deeply lying structures. On cutting into the limb the enlargement is seen to be due to great hypertrophy of the cutis vera and the subcutaneous tissue, which together may have a thickness of one to many inches. To a less extent the intermuscular areolar tissue and fasciæ share in this hypertrophy, and in rare cases even the periosteum and bones. The epidermis is not uniformly affected and often is unchanged, but sometimes patches of it show thickening of the Malpighian layer. Irregularly distributed strands of fibrous tissue are found in the thickened dermal and subdermal layers, which add to the firmness of the overgrowth. The muscles show no change or are wasted from pressure and partial disuse. The glands and hair follicles are more or less degenerated. The lymphatics are much dilated, prominent, and distended.

Etiology.

Elephantiasis in its endemic form is extremely prevalent in tropical and semi-tropical countries. Low-lying swampy districts where the water is stagnant favor the disease more than those which are higher and well drained. Mere altitude, however, would not seem to be an etiological factor, for the disease is common in mountainous districts, such as those of Samoa and Madagascar among many others. In the endemic areas the disease falls much more severely on the natives than on the resident Europeans. In India Sir Joseph Fayrer thinks this is due to racial differences and says that Europeans who are affected nearly always have some admixture of dark blood in their veins. On the other hand, Manson thinks the explanation lies in differences of opportunity, that the European by his habits of life is less exposed to the exciting causes of the disease, does less manual labor, and is protected by his clothing from those small injuries which are

the immediate cause of the attacks of lymphangitis; and, moreover, he is more careful of the water he drinks and of the food he eats. As showing how exposure to slight injuries, by causing lymphangitis, may determine the outburst of the disease, Manson mentions his experience that, taking the natives, elephantiasis of the legs is more prevalent among the laboring classes than among the well-to-do, whereas no such difference is observable in elephantiasis of the scrotum or chyluria. In support of this view he notes also the fact that when Europeans assimilate their modes of life to those of the natives they become equally liable to attack.

Women are less often affected than men, probably because they are less exposed to attacks of lymphangitis.

The affection is most common in young adult and middle life and does not frequently commence in childhood or old age.

In the production of sporadic elephantiasis climate and race play no part. Any condition which impedes the efferent flow of lymph and, of less importance, of blood from a part prepares the way for elephantiasis. The impediment may be central or local to the part affected. Thus obstruction to the thoracic duct, compression of the efferent lymph trunks of a limb by tumor, bony outgrowths, or diffuse inflammatory thickening, and enlargement and occlusion of the lymph glands have all led to elephantiasis. But here, too, the immediate cause of the hypertrophy is recurring attacks of lymphangitis and deep dermatitis, probably excited by slight injuries acting on a lymph-stagnated area. Lastly elephantiasis is sometimes congenital.

Symptoms.

The endemic form first manifests itself by an attack of elephantoid fever, the constitutional symptoms of which closely resemble an attack of erysipelas, viz., headache, malaise, nausea, and vomiting. Accompanying the general symptoms the scrotum, leg, or arm, as the case may be, becomes inflamed, swollen, tense, and tender. Vesicles may temporarily form on the surface of the swollen limb and give escape to lymph, with corresponding relief of tension. The general symptoms subside after a period varying from a few hours to a few days, and their subsidence is followed by a slow abatement of the local swelling and tenderness. One such attack forebodes others, which recur with varying severity at intervals of days, weeks, or months. Between whiles there is perfect health. The constitutional disturbance may be very marked or so slight as to be hardly noticeable. After each attack of inflammation, the permanent enlargement and deformity of the affected parts becomes greater, the size being

proportional to the number and severity of the attacks. With the lapse of years the latter commonly become slighter and less frequent and ultimately cease, or are so slight as to give rise to no symptoms. The growth of the affected part is not always arrested by the cessation of the lymphangitis but may continue notwithstanding, albeit at a slower rate. The general health in no way suffers; what trouble there is arises from the impediment to free movement and the liability to injury offered by the tumor. Although all regions of the body can be affected by elephantiasis the disease shows a marked preference for certain localities. In the vast majority of cases the legs are the seat, alone or in company with other parts. Next in order of frequency come the upper extremities and the scrotum, and other regions such as the scalp, face, and female genitals now and again contribute examples. In endemic elephantiasis of the lower extremities the brunt of the disease usually falls upon the legs, ankles, and feet, and the thighs are free or only slightly affected. The leg and foot become one distorted mass, with a constriction at the level of the ankle, although the outlines of the latter are quite lost; it is no uncommon thing for the calf to measure thirty inches in circumference. The inguinal glands are variously affected and during the attacks of elephantoid fever there is always some adenitis. Some authors note a considerable enlargement of the glands, which may sometimes be bilateral although the elephantiasis is confined to one leg. Others, like Turner, who speaks from wide experience in Samoa, describe only an enlargement of the glands, during the attacks of lymphangitis, of a slight and transitory character.

Elephantiasis of the Scrotum.—These scrotal tumors attain a great size and commonly weigh about ten pounds. Much higher weights have, however, frequently been recorded and masses of one hundred, and even on one occasion of two hundred and twenty-four, pounds have been removed. Hydroceles are often present and contribute to the weights of the tumors. The skin is indurated, rough, and wrinkled, and, as is the case elsewhere, its sensitiveness is diminished. The penis is hidden from view and lies at the end of a long canal which opens on the anterior surface of the tumor. Lymph scrotum is a frequent antecedent to elephantiasis, the former condition merging into the latter.

Sporadic elephantiasis like the endemic form most frequently affects the legs, and for the same reason, viz., that return of lymph is, owing to their position, more difficult here than elsewhere, and accordingly even a slight obstacle in the course of the lymph tracts makes itself easily felt. The enlargement of the limb is more uniformly distributed in this than in the tropical variety, and is not so frequently limited to

the part below the knee. The history of such a case consists of recurring attacks of cellulitis, each leaving behind it a slight permanent addition to the size of the limb. The constitutional symptoms accompanying these attacks vary in intensity but are not usually severe. Among the causes which produce the original impediments to lymph flow may be mentioned tumors pressing upon the efferent lymphatics and veins of the limb, an acute attack of cellulitis with much effusion, which produces tension and pressure in surrounding parts, and phlegmasia alba dolens, especially where there is valvular disease. A syphilitic inflammation of the lymphatics leading to elephantiasis has been described.

Pathology.

In its causation elephantiasis is intimately related to the lymphangiomata and ectasias produced by occlusion of lymph tracts. The lymph vessels of the affected parts are not only found dilated and distended, but there is often escape of lymph or chyle from superficial blebs or from well-defined groups of vesicles. It follows, accompanies, or precedes ectasias, both internal and external, which are the result of obstruction of the thoracic duct or of other large lymph trunks. Thus chyluria, chylous ascites, or chylous discharges from the skin are all from time to time associated with elephantiasis in the same patient, and in non-filarial as well as filarial cases. Myers (Transactions of the International Medical Congress, Berlin, 1890) records a case of elephantiasis of the left lower extremity, with chyle-discharging vesicles on the left thigh, which vesicles subsequently dried up and gave place to chyluria. Quincke instances elephantiasic hypertrophy and œdema of both legs, accompanying chylous ascites. It is not uncommon for lymphangioma of the scrotum to gradually merge into elephantiasis, and patients who come under treatment for the latter affection often give a history of antecedent lymph scrotum. Further, elephantiasic hypertrophy may often be found supervening in the regions of the more circumscribed lymphangiomata. We see this in macroglossia, and in the interesting case recorded by Roberts, already quoted, of external lymphorrhœa complicated by chyluria, in which the cutis and subcutaneous tissues of the abdominal wall, where the lymphangioma was situated, showed great hypertrophy, measuring together from a half to one inch in thickness. Attempts to cure lymph varices, *e.g.*, lymph scrotum, and varicose inguinal glands have resulted in the setting up of elephantiasis. This fact is explained by supposing that the collateral circulation of the lymph is interfered with by these proceedings and that the resulting impediment leads to raised lymph pres-

sure, retarded flow, and formation of new varices, and at the same time it suggests that the conditions preceding and following the treatment are correlated. It is quite certain then that the first step in the production of elephantiasis is occlusion of the lymph channels. The occlusion may be situated either centrally in the thoracic duct or locally in the efferent lymph channels of the affected part. In the former case the dilated vessels of the elephantiasic area will be part of a more general ectasia and the stagnation of the lymph will be partial, the degree depending on the efficiency of the anastomotic circulation. In the latter case the lymph varix will be confined to the affected part, and the degree of stagnation of lymph will vary with the extent of the obstruction and the establishment or not of anastomoses; in some instances it is partial while in others it is absolute and complete. In the former again, the cause of the elephantiasis may be any one of those enumerated above as efficient in the production of thoracic-duct obstruction, such as compression by tumor, obliteration of the lumen by a parent filaria, etc.; and in the latter, the cause of the stasis may be pressure from without, congenital occlusion, or blocking of the vessels and glands by filarial ova.

Owing to the work of Dr. Patrick Manson * it is now an established fact that endemic elephantiasis is frequently (further investigation may show it to be invariably) a filarial disease. Its frequent association with admitted manifestations of filariasis in the same districts and even in the same individuals, and the fact that lymph varix and stasis are among its constant features, strongly point to this conclusion. The hesitancy shown by many authorities to accept this view has arisen from the fact that, whereas in tropical chyluria, chylocele, and the other filarial diseases, the presence of filariæ in the blood is the rule, in elephantiasis it is the exception. This admitted fact Manson explains by supposing that the parent filaria is lying in a lymphatic area which is completely occluded; that in elephantiasis of the lower extremity, for instance, in which the varix is local and not part of a general ectasia, the parent worm is situated in the lymphatics on the distal side of the groin glands; that its embryos or ova, though present in the lymph of the limb, cannot reach the blood stream on account of this occlusion of all the lymphatic outlets. To explain the total occlusion, Manson throws out the suggestion that the female worm is aborting—that instead of normal embryos it is discharging into the lymph stream immature ova, which, in virtue of their larger diameter, their inelastic envelopes, and their incapability of movement,

* Vide Article "Filarial Diseases" by that author in Davidson's "Diseases of Warm Climates," to which the writer is much indebted.

become impacted in the lymph glands, and that the latter slowly, one by one, become blocked until the lymphatics of the limb are entirely shut off from those of the body generally. That the parent filaria does sometimes thus abort has been conclusively shown by many observations. We may regard it as established then that endemic elephantiasis is often a filarial disease, but it is still an open question whether it is invariably so. Dr. Turner (M'Call Anderson's "Diseases of the Skin"), speaking from an extensive experience in Samoa, expresses himself against its filarial origin, and in favor of its relationship to malaria. In support of his opinion he mentions that though elephantiasis is very common in the island, lymph scrotum and chyluria are unknown; that the disease is particularly prone to make its appearance after exposure at night; that quitting the endemic area often leads to recovery, and that quinine is an efficient remedy. The presence or absence of filaria nocturna in Samoa has not yet been determined. The first step in the production of elephantiasis is accordingly occlusion of lymphatic vessels, and in the endemic form the cause of the occlusion must in many cases be the filaria nocturna.

At the same time it should be clearly understood that mere occlusion of lymphatic vessels, provided a collateral circulation is established, will not produce elephantiasis or even disturb the health. But although a condition not directly productive of harm, it is a constant source of danger; an occlusion which is partial to-day may become complete to-morrow, and a limb in which there is even a partial impediment to lymph flow is more vulnerable than one in which the flow of lymph is quite free. Very slight causes would serve to light up, in a limb thus predisposed, the attacks of cellulitis which mark the commencement and the progress of the hypertrophy. Owing to the impaired circulation the effused products are only imperfectly absorbed, and after each recurrence of inflammation there is a permanent addition to the size of the limb. Furthermore, with the progress of the hypertrophy, the circulation becomes more sluggish and increase in the demands made upon it goes hand in hand with decrease in its efficiency.

Congenital elephantiasis resembles the acquired form in its anatomical characters. Many examples of this condition have been recorded, and it is exemplified by macroglossia, the structure and causation of which have already been described.

Treatment.

During the acute attacks of lymphangitis, rest in bed, elevation of the part, and application of evaporating lotions are indicated, with a saline mixture internally. For elephantiasis of the leg, rest, elevation of the limb, elastic bandaging, and massage comprise the routine treatment; these measures palliate, although they cannot cure the disease. Electricity, in the forms of galvanism and electrolysis, has met with success in some hands; the former is applied for fifteen to twenty minutes at a time with the negative pole on an affected and the positive pole on an unaffected area. Ligature of the femoral or external iliac arteries has been performed for both the endemic and sporadic affections, but in neither case have the results been encouraging. In very extreme degrees of hypertrophy amputation of the limb in rare instances has become necessary. For elephantiasis of the scrotum, if the tumor be large and growing, complete removal is practised and with brilliant results. The details of this surgical procedure have no place in a work on medicine. Those who believe that elephantiasis is related to malaria recommend removal from the endemic area and the internal administration of quinine and arsenic.

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DISEASES

OF THE

THYROID GLAND.

INCLUDING

MYXŒDEMA, CRETINISM, EXOPHTHALMIC GOITRE, GOITRE, ETC.

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DISEASES OF THE THYROID GLAND.

THE subject of diseases of the thyroid gland belongs partly to medicine and partly to surgery. The greater part of this article is devoted to the consideration of the medical diseases of the thyroid gland which are accompanied by general symptoms, namely, myxœdema, cretinism, and exophthalmic goitre, as they are of greater interest to the physician. The local diseases of the thyroid gland which do not cause constitutional disturbance are considered more briefly, as they frequently require surgical rather than medical treatment.

MYXŒDEMA.

(*μύξα*, mucus; and *οἴδημα*, a swelling.)

Synonyms.—A cretinoid state supervening in adult life in women (Gull); *cachexie pachydermique* (Charcot); *cachexia thyroidea, cachexia strumipriva* or *thyreopriva* (Kocher).

DEFINITION.

Myxœdema is an affection characterized by widespread changes in nutrition as shown by the appearance of a solid œdematous swelling of the subcutaneous tissues, dryness of the skin and arrest of development of its appendages, subnormal temperature, slowness in mental processes and in the execution of voluntary movements. When it arises in early life an arrest of mental and bodily development occurs in addition to the symptoms observed in the adult. The condition is due to loss of function of the thyroid gland and the consequent diminution of the supply of the secretion of the gland to the blood. The severity of the symptoms depends upon the amount of injury which has been done to the secreting tissue of the gland, and not upon the nature of the process which has brought about the destruction of it, so that the same symptoms may follow fibrosis leading to atrophy, conversion into goitre, or removal by operation. Strictly speaking myxœdema is a symptom or rather a collection of symptoms of destruction of thyroid-gland tissue, just as ascites may be a symp-

tom of cirrhosis of the liver. It would be more exact to speak of the ordinary form of the disease as chronic interstitial thyroiditis or cirrhosis of the thyroid gland, for although myxœdema is curable by thyroid extract the fibrosis of the thyroid gland remains incurable. Lanz suggests that all forms of myxœdema should be termed *cachexia thyroidea*, when due to disease he suggests the name *cachexia thyreopriva*, and when the result of operation *cachexia thyroidectomica*. We shall, however, in this article use the terms myxœdema and cretinism in their usual acceptance.

History.

The history of myxœdema is a short one, for it is little more than twenty years since attention was first directed by Sir William Gull to the existence of this condition, the main features of which are now so well known. Though the history of this disease is short it is an eventful one, for in this short period we have acquired more knowledge of its nature and appropriate treatment than we yet possess in the case of some diseases, the symptoms of which have been well known for centuries.

During these twenty years clinical observations have furnished us with a full account of the symptoms as they occur in the various stages of the disease.

Post-mortem and microscopical examinations have revealed much of the morbid anatomical changes which underlie the symptoms and have paved the way for experimental research, which has thrown a great deal of light on the pathology of the condition and has indicated a rational means of treatment that has proved successful in removing the symptoms of this previously incurable malady.

We have no means of ascertaining how long myxœdema may have existed, as there is no description of it in medical writings before 1873. In that year Sir William Gull[1] first gave a clear description of the symptoms in a paper "On a Cretinoid State Supervening in Adult Life in Women." This paper was read before the Clinical Society of London and in it he described many of the characteristic features of the disease. He also drew attention to the similarity between this condition and cretinism as described by Fagge,[91] and expressed the opinion, which has since been proved to be correct, that the one was allied to the other.

In 1877 W. M. Ord[2] read a communication "On Myxœdema, a Term Proposed to be Applied to an Essential Condition in the 'Cretinoid' Affection Occasionally Observed in Middle-Aged Women," before the Royal Medical and Chirurgical Society of London.

Ord there described the clinical features of five cases and the results of a post-mortem examination made on one of them by W. S. Greenfield, and of a chemical examination of some of the swollen skin by Cranstoun Charles. The relationship of the condition to endemic and sporadic cretinism was discussed, and as a result of his observations Ord suggested that the symptoms were due to the jelly-like swelling of the connective tissues. As pieces of the œdematous skin yielded an excess of mucin, this mucous œdema was considered to be the most characteristic feature of the condition and he proposed for it the name myxœdema, which has since then been accepted as the usual designation for this disease.

In the same paper the altered condition of the thyroid gland was described, and a microscopical specimen was figured in which the proper glandular structure was shown to be almost entirely replaced by fibrous tissue.

After this, cases were recorded by different observers in different countries. Among others may be mentioned Charcot,[3] who in 1881 gave the result of his clinical observations and proposed the name "cachexie pachydermique" for the disease. The following year Jacques L. Reverdin[4] described a chain of symptoms which he had observed in cases after total removal of the thyroid gland for goitrous enlargement.

These symptoms were the same as those which had been already observed in cases of myxœdema in England, though at that time no mention was made of the similarity which existed between the two conditions.

In 1883 Kocher[5] also described a similar condition, which had appeared in patients from whom he had entirely removed goitrous thyroid glands, as "cachexia strumipriva." He, however, did not attribute the condition which followed the operation to loss of the thyroid gland, but believed that it was produced by a chronic asphyxia which occurred as a result of injuries received by the structures in the neck during the removal of the goitre. Two months later Reverdin discussed the relationship between myxœdema and cachexia strumipriva. A few months afterward Felix Semon,[6] being struck by the identity of the symptoms which occurred in myxœdema and in cachexia strumipriva, suggested that the loss of function of the thyroid gland, occurring as it did in both conditions, was probably the common factor in the production of each of them.

At this stage a most important investigation was undertaken by a special committee appointed by the Clinical Society of London on December 14th, 1883, in order to gain further information of the subject of myxœdema and its relationship to the allied conditions of

cachexia strumipriva and cretinism. The report of this committee contains a great deal of valuable information and is the most extensive account of the disease which has yet been published. As a part of this investigation Victor Horsley undertook a series of experiments on monkeys by means of which he was able to show that cachexia strumipriva was due to the loss of function resulting from removal of the thyroid gland. The report of the Clinical Society's committee[6] also contains the clinical record of one hundred and nine cases of myxœdema, accounts of the morbid anatomy of the disease with chemical investigations of the diseased tissues, and the results of partial and total removal of the diseased gland in man. The general conclusions of the committee based on all these observations were to the effect that: 1. Myxœdema is identical with cachexia strumipriva; 2. Sporadic cretinism is myxœdema occurring in childhood; 3. Endemic cretinism is also closely allied to myxœdema; and further, that while these conditions are dependent on loss of function due to removal or disease of the thyroid gland, the ultimate cause of this loss of function in ordinary myxœdema is not as yet explained. Valuable observations upon the symptoms and morbid anatomy were published by Hun and Prudden.[7] Byrom Bramwell[8] has published some excellent and lifelike illustrations of the disease in his "Atlas of Clinical Medicine."

The next advances were in the direction of rational treatment. Von Eiselsberg[60] found that the thyroid gland could be transplanted from the necks of animals to some other part of the body so as to continue its functions and prevent the onset of cachexia strumipriva. Quite independently, and from his own observations, Victor Horsley was led to suggest in 1890 the grafting of a sheep's thyroid gland into a patient suffering from myxœdema as a means of arresting the progress of the disease. This suggestion was acted upon by Bettencourt and Serrano,[10] of Lisbon, with marked benefit to the patient commencing directly after the operation. From this they concluded the benefit was derived from absorption of the juice of the thyroid gland. This observation led the author[11] to suggest in 1891 the employment of a thyroid gland extract as a remedy for the disease, the efficiency of which he[12] was soon afterward able to prove by injecting the remedy hypodermically in a well-marked case of myxœdema. In 1892 it was shown by Howitz,[13] of Copenhagen, and soon afterwards by E. L. Fox[14] and by H. Mackenzie[15] in England that the beneficial effects of the remedy which have followed its employment by hypodermic injection could be obtained by the simpler method of giving the gland itself, or some preparation of it, by the mouth.

During the last four years many papers have appeared describing the beneficial results to be obtained by the administration of the thyroid gland itself and of various preparations made from it. These articles contain many valuable observations on the disease, the means of administering the remedy, and the effect produced by it.

Varieties.

The varieties of myxœdema may for the purposes of description be most conveniently divided into two groups: firstly, those in which the condition occurs as a result of destruction of thyroid-gland tissue by disease; and secondly, those in which it is the result of the removal of the gland by operation.

In the first group there are several varieties of myxœdema which differ in some respects from the typical adult form of the disease that we are accustomed to see in middle-aged women and men. The differences between these varieties depend upon the age at which the disease first appeared and the nature of the process which led to the diminished functional activity of the thyroid gland. In this connection it must be borne in mind that the actual size of the thyroid gland is not necessarily an indication of the amount of functional activity of which it is capable. A considerably enlarged gland may, from the nature of the structural enlargement, be just as incapable of carrying on its normal function as an atrophied gland.

The most remarkable variety is the infantile form, which is congenital or commences in early childhood. The condition which develops under these circumstances is known as cretinism, and is characterized by an almost complete arrest of mental and bodily development in addition to the usual symptoms of the disease as observed in the adult. Cretinism occurs endemically in certain districts of some countries and then is not infrequently complicated by the presence of a goitre, such cases being known as goitrous cretins, the enlargement of the thyroid gland being associated with loss of its function. The sporadic form of cretinism is associated with absence or atrophy of the thyroid gland just as in the adult form of the disease. These varieties will be considered separately from the adult form on account of the many special features which they possess.

Intermediate between the infantile and adult forms of myxœdema are the somewhat rare cases in which the disease has commenced at some age between infancy and puberty. In these the symptoms are the same as in the adult, but there is in addition arrest of development at the stage at which it had arrived when the symptoms first appeared, so that we find all varieties intermediate between infantile cretinism

and adult myxœdema. The earlier the onset the more nearly the symptoms conform to the type of cretinism, and the later the onset the nearer the resemblance to myxœdema in the adult. All those, however, in whom the disease has commenced before the age of fifteen we shall consider as cretins.

Of the adult form there is a slight variety which I have observed several times in women between forty and fifty years of age. In this the symptoms are very slight and as far as my observation goes they do not tend to get worse, in fact I believe that they may recover spontaneously, either because the thyroid gland regains some of its lost activity or else it may be that the part of the gland which is left still functionally active is sufficient to maintain health again as age advances and the thyroid becomes of less importance.

In each variety we find varying degrees of severity. There are cases in which the symptoms are slight and at times by no means easily detected. These slight cases are often in the early stage and may become more severe as time goes on. All degrees of severity have been observed from these slight cases to those in which the disease has rendered the patient totally incapable of mental or bodily effort.

In the second group in which the symptoms are due to the removal of the thyroid gland by operation we also find different types which depend upon the age at which the thyroid gland was removed. In the adult form the symptoms are identical with those of ordinary myxœdema. In cases in which the thyroid gland has been removed in childhood the symptoms have been the same as those of sporadic cretinism. The first example of this type was described by Paul Bruns,[96] in which the patient, eighteen years after thyroidectomy had been performed at the age of ten, presented the characteristic features of a cretin.

Etiology.

Of the actual exciting cause of the changes in the thyroid gland which lead to the development of myxœdema, in the great majority of cases we as yet know nothing. In a few cases the destructive lesion of the thyroid gland has been traced to a definite exciting cause. Köhler has recorded two cases in one of which syphilis[16] and in the other actinomycosis[17] of the thyroid gland led to the development of myxœdema. There are certain conditions under which the disease most frequently develops and which may be considered as predisposing causes, and as such require consideration in detail.

Age.—Myxœdema may develop at any period of life from infancy to old age, but when the disease occurs before full development has

taken place it has special features, so that we shall here consider only the adult form of the disease commencing after the age of fifteen. Hun and Prudden[7] found that the average age at which the disease commenced in 76 women was thirty-eight years and in 20 men forty-two years. The following table compiled from 274 cases in which the age at which the symptoms were first noticed was recorded, shows the comparative frequency of onset in each period of five years from fifteen to eighty.

	Males.	Females.	Total.
Between 15 and 20	1	6	7
" 20 " 25	1	19	20
" 25 " 30	3	18	21
" 30 " 35	0	31	31
" 35 " 40	8	42	50
" 40 " 45	7	45	52
" 45 " 50	6	28	34
" 50 " 55	3	23	26
" 55 " 60	1	18	19
" 60 " 70	0	10	10
" 70 " 80	1	1	2
Total	31	243	274

From this table we see that in women myxœdema may occur at any time of life, from fifteen years of age up to seventy. From fifteen to forty-five there is an increasing number in each succeeding period of five years, after which the numbers diminish. Nearly all the cases, however, occur between twenty and sixty and more than half of the total number between thirty and fifty. In comparing the different periods with each other we find that the disease develops most frequently between thirty-five and forty-five and rather more frequently in the five years following the fortieth year than in the five years which precede it. Thus it is evident that between thirty-five and forty-five and especially between forty and forty-five a woman is more liable to myxœdema than during any other similar period of her life. In men also the disease may occur at any age from fifteen to seventy, but in two-thirds of the cases the disease first appeared between thirty-five and fifty, so that this is evidently the period of life during which the disease is most liable to develop.

Sex.—Myxœdema occurs very much more frequently in women than in men, so that sex plays an important part in predisposition to the disease. Hun and Prudden[7] found that out of 150 cases 113 were females, 32 males, and the sex was not stated in the remaining 5. In a series of 370 cases of which I have been able to ascertain the sex, 322 were women and 48 were men, showing that the disease is nearly seven times as frequent in the female as it is in the male.

Heredity.—In some families there is a distinct predisposition to disease of the thyroid gland. Several members of the same family may be affected by the same disease or different forms of thyroid disease may appear in members of the same family. Of the latter Maude has recorded an example in the case of a family in which the mother was myxœdematous and the daughter suffered from exophthalmic goitre. Myxœdema does occasionally occur in more than one member of the same family.

The Clinical Society's report mentions one family in which three sisters all developed myxœdema. Hun and Prudden[7] mention one family in which three sisters were myxœdematous, one of whom had a daughter who showed early signs of the disease; one in which both the father and sister of the patient had myxœdema; two families in which two sisters, and two others in which both mother and daughter were affected. I have information of one family in which two sisters were myxœdematous, and another has been observed by Mitchell and Craig in which a brother and sister were both affected. Putnam[18] records a case in which a cousin and an aunt also had myxœdema.

From these examples it will be evident that in a few cases the predisposition to the disease is inherited from one of the parents and that in some families there is a tendency for the disease to develop in two or three members of the same generation of the family. Of the other diseases which appear in the family histories of patients suffering from myxœdema the most frequent are tuberculosis and functional diseases of the nervous system, so that they may possibly play some part in producing a predisposition to the disease.

Locality.—The distribution of myxœdema is a question of great interest, and a fuller knowledge of this subject may throw light on the part which local conditions appear to play in the production of the disease. The data we now possess show that the disease occurs much more frequently in some parts of the world than in others. It rarely develops in the tropics.

One case is recorded by Bramwell[8] in which the symptoms developed in India in the district of Rajputana, and another by Wilson[19] in which the disease appeared after more than twenty-five years' residence at the Cape of Good Hope.

Cases of myxœdema are not very rare in Australia and as many as nineteen cases were recorded in a recent number of the *Australasian Medical Gazette.*[20] Cummins[21] has observed two cases in males in Thibet. I know of no record of any case of myxœdema which has been observed in any of the colored races. Whether this fact depends upon climate or race, or the ease with which a case occurring in a colored woman might be overlooked, I am unable to say. Myx-

œdema certainly occurs most frequently in cold climates and is probably more frequent in Europe than in any other continent.

The disease is well known in America, but it does not appear to be so common there as it is in Europe. Of European countries more cases have been recorded in the British Isles, where the disease was first described, than in any other country. The distribution of the disease in Great Britain is by no means uniform, as cases are not uncommon in some parts of the country, while in others they are much less frequently seen. In the Clinical Society's report the residence of 76 of the patients is stated; of these 46 lived in England, 5 in Scotland, and 2 in Ireland. These figures do not at all represent the number of cases which have been recently recorded in these different countries, for from Scotland alone more than 50 cases have been reported during the last two years. In Edinburgh and Dundee especially, myxœdema is not at all uncommon. In a recent discussion at the Edinburgh Medico-Chirurgical Society[22] Byrom Bramwell mentioned that in Edinburgh (population 261,261) alone there were some 25 or 30 cases of myxœdema and cretinism, a considerable portion of which belonged to a particular district of the city, and Stalker stated that in Dundee (population 153,066) he knew of at least 21 cases.

In England myxœdema appears to be more prevalent in the north than in other parts of the country, and especially in the lower parts of the Tyne valley which runs for a distance of some ten miles from the west end of Newcastle to the German ocean. On the banks of the Tyne in the towns of Newcastle, Gateshead, Felling, and South Shields, which have an aggregate population of 368,000, during the last three years I have either seen or been informed of 29 cases of myxœdema. Though not so common here as in Edinburgh and Dundee, I do not know of any other district in England where there are so many cases in proportion to the number of inhabitants.

In Ireland I have information regarding 6 cases, and Bramwell[8] mentions 3 others. In Lower Brittany Morvan[23] reported 11 cases, otherwise the disease is not common in France. In Germany and Belgium myxœdema is rare, and in Norway[25] only 4 cases have been recorded.

These facts show that in certain districts and towns myxœdema is endemic, but we do not as yet know what special conditions in these places are responsible for the greater frequency of the disease. Social position does not appear to have any special influence upon its production, as cases occur among all classes of society. The larger number come from the lower class, but this is fully accounted for by the larger numbers which compose this class.

Personal Antecedents.—Neither syphilis nor alcohol appears to

cause any special predisposition to myxœdema. It is an interesting fact that in some cases of myxœdema the onset of the disease is preceded by enlargement of the thyroid gland accompanied by other symptoms of exophthalmic goitre. In these cases it seems very probable that the myxœdema was a late result of changes which started in the thyroid gland during the attack of exophthalmic goitre and ultimately led to atrophy. The very marked excess of cases of myxœdema among women naturally raises the question of the association of the onset of the disease with changes in the female generative system. Variations in size of the thyroid gland are not infrequently noticed in connection with menstruation and pregnancy. The larger number of cases of myxœdema occur in married women who have borne children. Thus of the cases collected by the Clinical Society 69 were married, only 7 of whom had not borne children, 14 were single, and 1 a prostitute.

Of 113 cases occurring in women collected by Hun and Prudden,[7] 82 were married, 14 were single, and in 17 this point is not stated. Out of 64 of these cases in which a statement is made as to the family, only 4 had not borne children, and the other 60 had more than 300 children and 29 miscarriages. Thus excessive child-bearing seems to be a predisposing cause to myxœdema. Excessive uterine hemorrhage is also said to be a frequent antecedent, but as hemorrhage is a common symptom of myxœdema itself, this may in such cases be an early symptom rather than a predisposing cause. It should also be pointed out that the greater number of cases of myxœdema in women commence between forty and forty-five years of age, so that in these cases the functional activity of the thyroid gland first begins to show signs of failure about the same time of life that the female generative system also ceases to be productive. It is not unreasonable to suppose that the atrophy of the former may in some way be connected with the menopause, though we do not often find it stated that the disease commenced just at that time.

Symptoms.

The symptoms of myxœdema develop slowly, and the onset is so gradual in the great majority of cases that it is often difficult to ascertain how long the disease has lasted. It not infrequently happens that several months or even a year may elapse before the symptoms become really distinct. Occasionally they develop more rapidly and become well marked in the course of a few weeks, so that within two months from the commencement of ill health the symptoms are sufficiently well marked to suggest the nature of the disease

at the first glance. This rapid development occurs more frequently when the disease commences in young adults than in those of more advanced years.

At the commencement of the disease the patient may at first only be conscious of a certain degree of languor to which she is unaccustomed. The ordinary round of household duties which used to be accomplished without conscious effort begins to be a burden and much more energy is required in doing work of any kind than was necessary before. Some who are naturally energetic struggle against this languor, while others soon give way to it, give up most of their work, and stay indoors as much as possible. In winter the cold is felt more acutely than in former years, and the patients complain that they cannot keep warm and consequently sit as near as possible to the fire. In addition to disinclination for effort there is loss of activity so that all voluntary movements are performed more slowly than usual. A change in the appearance is noticed, especially by those who have not seen the patient for some time, owing to the gradual accumulation of the myxœdematous swelling in the subcutaneous tissues of the face. This gradual change being accompanied by general increase in the size and weight of the body is often mistaken for ordinary obesity. In these early stages of the disease it is not always easy to come to a conclusion as to the true nature of the case, for the more profound changes in nutrition which are so well marked in a case of longer duration are absent in the early stages. Thus the skin may remain fairly moist, the hair as plentiful as ever, and the temperature at the normal level. In a fully developed case of myxœdema the symptoms of the disease are very characteristic and require description in detail.

Solid Œdema.

The most characteristic symptom of myxœdema, and that from which the disease takes its name, is the solid œdema which develops in the subcutaneous tissues of the body. This swelling is most fully developed in the loose subcutaneous tissues and is scanty in parts where the skin is firmly fixed to the structures which lie beneath it. Thus, for example, it is generally abundant on the back of the hand, while little can be detected beneath the skin of the palm. The swelling is in most cases first of all detected in the face, as it produces a very considerable alteration in the appearance of the patient. In some cases it is swelling of some part of the upper or lower limbs which first attracts attention, in which case the lower limbs appear to be more frequently the seat of the early swelling than the upper. Sooner or later a general solid œdema is developed. Although the

swelling resembles a watery œdema in appearance, such as is seen in Bright's disease, on pressure with the finger it is found to be solid and no depression is left at the point where pressure has been made. If the swollen part is pricked, no fluid exudes unless the solid œdema is associated with watery œdema as well, as is the case sometimes in the lower extremities. When the swelling is well developed it may vary in amount from time to time. The changes produced in facial expression by the swelling are often very remarkable, and so great is the alteration in some cases that the patient is with difficulty recog-

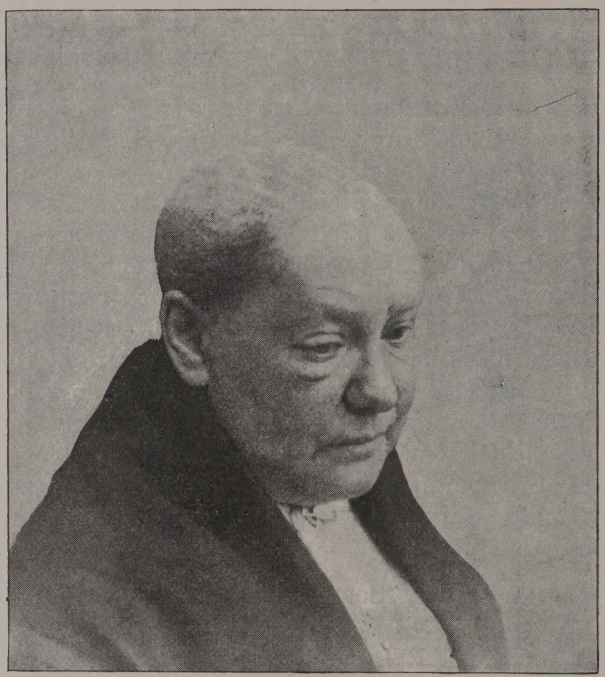

FIG. 44.—A Case of Myxœdema in a Woman Aged Fifty-two Years. Duration of the disease, twelve years.

nized by friends who have not seen her since the commencement of the disease. The swelling tends to obliterate the special character of the individual features and to smooth out the natural wrinkles, the result being that many cases of myxœdema develop a family likeness to a common type. The general facial characters are well shown in Figs. 44 and 46. The swelling is well marked in the subcutaneous tissues of the upper and lower eyelids. The upper eyelid is wrinkled and the supraorbital fossa is filled up by the myxœdematous swelling. The skin just beneath the lower eyelid is swollen and pendulous and

looks translucent as if distended by a clear fluid or a gelatinous substance. As a result of the swelling of the upper eyelid it tends to droop and a difficulty is often experienced in looking upward. This obstruction to the upward view is in a measure compensated for by an overaction of the occipito-frontalis muscle, by the tonic contraction of which the eyebrows are kept constantly elevated. This contraction of the occipito-frontalis muscle causes the very marked transverse wrinkling of the forehead which is often seen in cases of myxœdema. The nose becomes thickened and enlarged, owing to the swelling of

FIG. 45.—The Patient Represented in Fig. 44, after Seven Months' Treatment by Thyroid Extract.

the subcutaneous tissues. The enlargement thus materially differs from that which takes place in acromegaly in which the actual structure of the nose itself is increased in size. In one case I have observed a deposit of golden-yellow fat under the exposed part of the orbital conjunctiva, which did not extend to the parts of the eyeball, which remained covered by the eyelids when the eyes were open. The cheeks are full and round and in some cases rather pendulous. The centre is occupied by a circumscribed pink flush. The lips are thick and everted and the mouth appears to be widened. There is often a

considerable amount of swelling of the subcutaneous tissues beneath the chin, and the neck is considerably increased in circumference so that a collar which fitted easily before can no longer be worn. The ears are often swollen and enlarged. The result of these changes in the features is that the face has a stolid expression, which varies but little with passing emotions as the natural lines and wrinkles in the skin are obliterated. The actual change in appearance which is due to the swelling can be most readily appreciated by watching the effect of treatment by thyroid extract, under which the swelling rapidly disappears and the face soon regains its normal appearance. This

FIG. 46.—A Case of Myxœdema in a Man, Aged Forty-four Years.

contrast is well illustrated by Figs. 44, 45, 46, and 47. In the supraclavicular region there is generally some fulness and in some cases "fatty" tumors have been described in this part, such as are commonly found in cretins. When the swelling is general, the trunk and limbs are increased in size. In this case the arms and legs are uniformly swollen, and if the swelling is well marked the movements of the joints are limited by it. Thus the patient may no longer be able to reach the back of the neck to fasten the collar behind, or to stoop sufficiently to lace the boots while sitting. The hands are swollen, the swelling being more marked on the back of the hand where the skin

is loose than on the palmar aspect. The hand like the face loses its characteristic features and looks broad and "spade-like." The movements of the fingers are rendered difficult and slow by the swelling. The feet also are swollen and in some cases the enlargement is in part due to the presence of watery œdema which pits on pressure. The vulva may be swollen or of normal size. The myxœdematous swelling adds materially to the weight of the body and as it develops the weight increases. The actual increase in weight can be estimated more exactly by taking the amount of weight which is lost during treatment by thyroid extract than by comparing the weight taken before

FIG. 47.—Patient Represented in Fig. 46. After six weeks' treatment by thyroid extract.

the onset of the disease with that taken when the symptoms are fully developed, for it is very difficult to fix the actual date of onset. The loss of weight which takes place during the treatment shows that the œdema may add as much as one or even two stones (fourteen to twenty-eight pounds) to the natural weight of the body.

THYROID GLAND.

In healthy persons it is often very difficult to make out the exact size of the thyroid gland by palpation and to determine if there is any

diminution in it. This difficulty is much greater if the size of the neck is increased by either subcutaneous fat or myxœdematous swelling, so that in a considerable number of cases of myxœdema the thyroid gland cannot be felt at all. When the size of the gland can be made out, it is in most cases found to be smaller than usual. Enlargement is rare. In the Clinical Society's report the condition of the gland was noted in 59 cases. In 22 it was found to be small or atrophied; in 3 of these there had been a previous enlargement. In 23 the gland either could not be felt at all or the condition was uncertain. In 4 cases there was enlargement of the gland, in 9 it was said to be normal in size. Hun and Prudden[7] found that the gland was diminished in size in seventy-eight per cent. of the cases tabulated by them.

Cutaneous System.

Skin.—In fully developed myxœdema, the skin and its appendages undergo very profound changes which become more and more marked as time goes on. The skin is dry and rough, and the superficial layers of the epidermis are often shed as a fine white powder. The skin of the face is generally more or less distended by the solid œdema. It is often yellow in color, and so marked may this discoloration become that, in one of my cases, the patient's friends thought he was suffering from jaundice. In the centre of each cheek there is a circumscribed pink flush due to dilatation of the small veins in the skin. The swelling of the face, the discoloration of the skin, and the central flush on the cheek together produce such a characteristic appearance that once it has been seen it can never be forgotten. This characteristic appearance is remarkably well represented in Bramwell's colored plates.[8] The skin of the backs of the hands is often much wrinkled unless it is distended by myxœdematous swelling. On the palms it is dry, rough, and not infrequently cracked. The skin of the feet is like that of the hands. In advanced cases perspiration is entirely absent and often has not been seen for several years. Sebaceous secretion is also absent. In the early stages perspiration is still present and the skin may remain fairly moist. Warty growths and moles are frequently present on the skin. Hun and Prudden[7] found they were present in twelve of the cases which they collected.

Hair.—The hair becomes thin and scanty and is almost lost in a large number of cases. This occurred in eighty-six per cent. of Hun and Prudden's cases. The loss of hair is most noticeable on the scalp. The hair may only be thin or the head may be quite bald, except at one or two points where a few straggling hairs may still

remain. The skin of the scalp under these circumstances becomes dry, scaly, and brown in color, with many cracks extending through the superficial layers of the epidermis (*vide* Fig. 44). The hair which does not fall out becomes fine in texture and soft to the touch. The hair of the eyebrows, eyelashes, axillæ, and pubes may also be very scanty or even absent altogether.

Nails.—The nails are often discolored, grooved, or cracked.

Teeth.—The teeth in many cases are brittle and carious and many of them are lost.

Mucous Membranes.—The mucous membranes are pale and opaque. The lips are often of a peculiar dead bluish color.

Temperature.

One of the chief symptoms in myxœdema is the subnormal temperature. In the early stages of the disease the temperature may be normal or even slightly above normal. In more advanced stages patients complain that they feel the cold in winter much more acutely than they did formerly and that they can never get warm when the weather is cold. Such patients like to spend most of their time in winter sitting well wrapped up close to the fire. In warm weather they feel much better. The skin, especially of the extremities, feels cold to the touch. The temperature in fully developed cases is almost continuously below normal. In some it is not more than 1° or 2° F. below normal, in others it is much lower than this. In the Clinical Society's report ten cases are mentioned in which temperatures between 93° and 95° F. were recorded; in one 77° F. was registered shortly before death. Hun and Prudden[7] mention one case in which the temperature fell as low as 66° F. before death, the pulse being 20 and the respirations 12 a minute. It is important to remember that in these cases a temperature of 98° to 99° F. is a sign of fever; thus in a case in which the temperature has been subnormal a rise of temperature to the normal level may be the sign of the commencement of some complication such as phthisis, such a rise being equivalent to one of several degrees above normal in a healthy individual.

Nervous System.

In almost every case of myxœdema there is a marked derangement of the functions of the nervous system. In a few cases the symptoms are due to actual structural changes in the brain. In the great majority of cases the symptoms appear to be due to functional derangement only, as they rapidly disappear under treatment. The

symptoms are mainly characterized by slowness in the execution of all the functions of the nervous system.

Mental Symptoms.—The changes in intellect are apparent in the majority of cases by slowness in comprehending any new subject, in thinking, and in executing any new project. In some there is an unusual persistence in one line of thought or action. The memory becomes defective during the early stages of the disease, recent events being more readily forgotten than those which occurred before the disease commenced. In 46 out of 71 cases recorded in the Clinical Society's report the memory was impaired. Hun and Prudden[7] found that of 102 cases in which the mental condition was described, in more than one-half the mind was dull and sluggish, the memory was deficient in one-third, and in one-fifth there was insanity.

The temper is placid and equable in many cases, but in some there is marked irritability. Many patients who are placid as a rule are easily put out by any unusual circumstance and are apt to become confused in the presence of strangers. They feel acutely the change in their appearance and are very sensitive to any remark which is made about it. Agoraphobia is a marked symptom in some cases. These symptoms combine to make myxœdematous persons very fond of staying at home, and many of them rarely if ever leave the house unless obliged to do so. Sleep as a rule is good and there is often drowsiness in the daytime. In some cases sleep is disturbed by abnormal sensations or dreams. Hallucinations of both sight and hearing are frequent in the more advanced stages of the disease; thus a human being or an animal may be seen, a familiar voice may be heard, or a sound as if some one had opened the door and entered the room in which the patient is sitting. In some cases the patient is only conscious of seeing some ill-defined object flitting across the room but is unable to state exactly what it was. In others the hallucinations are extremely clear and distinct. For example, one of my patients told me that after the death of her father she distinctly saw him on several occasions walk into the room and sit down at her bedside, and on one occasion she heard him say distinctly "A—— hinny (a Northumbrian term of endearment), you are getting too fat."

Insanity.—Well-marked insanity occurs in some instances, and cases of myxœdema are to be found in many of our large asylums. The association between insanity and myxœdema was first pointed out by Savage.[26]

Frequently the peculiarities of temper and disposition which have been already described as occurring in many well-marked cases of myxœdema gradually become exaggerated till the patient is actually insane. Insanity in such cases may occur as either acute or chronic

mania, melancholia, or dementia. The consciousness of the altered appearance causes most patients to shun observation and to be very sensitive to any remarks made about them. This condition may pass into one of suspicion and so into the delirium of suspicion. The mental weakness gradually increases and there may be occasional outbursts of violence until finally dementia is developed, often in conjunction with great physical weakness. In addition to these symptoms, doubt, irritability, and exalted ideas occur in some cases. In the late stages of insanity of myxœdema convulsions not infrequently occur, ending in coma and death.

Subjective Sensations.—Subjective sensations which may affect either general or special sensation are frequently experienced by myxœdematous persons. Coldness is very often complained of, especially if the temperature of the air is low. Headache in the frontal or occipital region is not uncommon. Neuralgic pains may be felt at times in various parts of the body and abnormal sensations of pricking, numbness, and the like. Noises in the ears and giddiness are also of frequent occurrence. Several of these abnormal sensations may occur in the same case. Cutaneous sensation is said to be diminished in some cases; in the majority, however, it is unaltered. Retardation of sensory impulses has also been described in a good many cases, but this is a difficult point to investigate, as due allowance has to be made for the slowness of response to stimuli due to the hebetude of the patient.

Special Senses.—Sight is in many cases impaired, but no doubt this is often independent of the disease. The committee of the Clinical Society concluded that in a small proportion of cases "general diminution of acuteness of vision" and "watering of the eyes" may be considered as caused by the disease. Hearing was impaired in more than half of the cases in which note was made of this function. The deafness may be on one side only or on both, and from the fact of its frequent occurrence is evidently in many cases due to the myxœdema and not accidental. Taste and smell are also impaired in some cases, but not so frequently as hearing.

Motor Power.—In almost every recorded case of myxœdema it has been noted that voluntary movements are executed more slowly than in health. This retardation, which is partly due to the myxœdematous swelling of the skin overlying the joints, is apparent in almost every movement, and especially in walking. The slow, leisurely gait has been compared by Bramwell[1] to that of the hippopotamus. Slowness of speech is also a very constant symptom and is mentioned as being present in 100 out of 104 cases collected by the committee of the Clinical Society. The speech is slow and deliberate and the voice

may be hoarse and monotonous and thick in tone, the latter character in many cases being due to the thickness of the lips and the increased size of the tongue. Some write slowly and the handwriting may be defective, but as a rule, if the patient is an educated person, well expressed letters of considerable length can be written.

Muscular weakness of the limbs and neck is common so that the head falls forward till the chin is supported by the sternum. Some are liable to fall from a sudden "giving way of the knees." Paralysis occurs only as an accidental complication and contractures of the muscles of the limbs are rare. Incoördination is not infrequently observed in varying degrees, in some cases it has led to falls and injuries as a consequence. The superficial reflexes are occasionally diminished but they are rarely absent. As a rule the deep reflexes are unaltered, but in some cases they are diminished or delayed and in a small number they are absent; in a few they are increased. In one of my cases the exaggeration of the reflexes was due to lateral sclerosis of the spinal cord which in all probability occurred as an accidental complication.

Circulatory System.

Blood.—In myxœdema there is as a rule distinct anæmia which is shown by the pale color of the mucous membranes. Anæmia appears early but does not reach any great intensity. There may be both a diminution in the number of red blood corpuscles and a deficiency in hæmoglobin. Examinations of the blood have shown that the number of red corpuscles in a cubic millimetre may be reduced from between 4,000,000 and 5,000,000 (the average number in health) to between 3,000,000 and 4,000,000 as in one of Bramwell's cases in which they numbered 3,320,000. That this diminution is really due to the myxœdema is proved by the fact that the red corpuscles increase in number as the other symptoms of the disease disappear under the treatment by thyroid extract. This is illustrated by a case recorded by Putnam[18] in which the number of red corpuscles varied from 3,120,000 to 4,240,000 in each cubic millimetre; six years later, after the patient had been treated with thyroid gland preparation for six months, the blood was found to contain 5,700,000 red corpuscles to the cubic millimetre. In 23 cases in which an examination of the blood has been recorded, the blood was found to be normal in 7; in 10 the number of red corpuscles was decreased, in 4 the number of white corpuscles was increased, in 2 the red corpuscles were diminished and the white increased in number. Kraepelin[27] has observed a marked increase in the diameter of the red blood corpus-

cles in 3 cases of myxœdema in which the number of the corpuscles and the amount of hæmoglobin were apparently normal. Others describe the appearance of the corpuscles as being normal. Kraepelin also has found that the specific gravity of the blood and of the serum is increased. In a case recorded by J. J. Schmidt,[28] Benario found the different kinds of white corpuscles in the following proportion: Polynuclear cells 63 per cent., lymphocytes 24 per cent., mononuclear cells 5.7 per cent., transitional forms 6.8 per cent., eosinophile cells 1.5 per cent. The red corpuscles numbered 4,200,000 to the cubic millimetre and the hæmoglobin amounted to 80 per cent., so that the blood was practically normal.

Heart.— In a large number of cases of myxœdema, especially in the early stages of the disease, the heart is normal. Fifty-four per cent. of the cases recorded in the Clinical Society's report and forty per cent. of the cases collected by Hun and Prudden were found to be free from cardiac disease. In cases of long duration, however, the heart is not infrequently affected to a considerable extent. The action of the heart then becomes weak owing to degeneration of the heart muscle. This may be partly a result of the prolonged anæmia and the life of inactivity which many patients are compelled to lead.

Most frequently there is weakness of the heart sounds, and accentuation of the second sound has been observed. Hæmic murmurs are heard in some cases. Enlargement of the heart, lesions of the aortic and mitral valves occasionally occur, but are probably only accidental complications. The weakness of the heart due to degenerative changes in its muscular wall has attracted more attention since the introduction of the treatment of myxœdema by thyroid extract. Under this treatment the general condition improves more rapidly than the heart, which, during the early stages of the treatment, may be unable to bear the additional strain which is brought upon it by the renewed activity of the body. Under these circumstances unusual strain has in several cases led to the death of the patient from cardiac failure. In one such case recorded by John Thomson,[22] extensive fatty and fibroid degeneration of the heart was found at the post-mortem examination; two of my patients, who had symptoms of cardiac degeneration, also died suddenly as the result of an unusual effort.[29]

Arteries.—No special change in the arteries can, as a rule, be detected during life, though in a few cases atheroma and thickening of the arterial wall have been described.

Pulse.—The pulse is less frequent than in health, regular, slow, weak, and of low tension. High tension is rare. If either cardiac or renal disease is present the rhythm, force, and tension of the pulse

may be altered accordingly. The capillaries are frequently dilated on the cheeks and nose.

Hemorrhages.—Hemorrhage frequently takes place in myxœdema and it must be regarded as one of the more common symptoms. The bleeding most frequently occurs as menorrhagia or excessive loss of blood during parturition. In some cases the gums bleed very readily, especially after the extraction of teeth. Bleeding may also take place from the nose, throat, or bladder, and some patients bleed very freely after such slight injuries to the skin as the prick of a pin.

Digestive System.

The lips, as already described, share in the general swelling of the face. The teeth are generally in a bad condition, being irregular and discolored; many of them are broken and carious or have disappeared altogether. The gums may be pale from the anæmia; they are frequently swollen and may be soft, spongy, or ulcerated, and in some cases they bleed very readily. Some patients are troubled by a thick mucous discharge which runs out of the mouth at night during sleep on to the pillow. The tongue in quite half the cases in which its condition has been noted is swollen, and in some cases to a considerable degree. Exceptionally it may be cracked and sore. The uvula and soft palate are also not infrequently swollen. The swelling of the lips, tongue, uvula, and soft palate no doubt contribute largely to the production of the peculiar character of the speech so often observed in myxœdema. Digestion may be impaired owing to the want of good teeth, and the bowels are generally constipated. Piles are not uncommon.

Urinary System.

The urine may be quite normal, especially in the early stages of the disease. In well-marked cases the urine is often pale with a rather low specific gravity ranging from 1.008 to 1.018. In some cases the urine is found to contain albumin, as a rule, however, in small quantities. Albuminuria occurred at one time or another in twenty per cent. of the cases collected by Hun and Prudden. In a few cases the albumin is due to actual disease of the kidneys, and in such the urine may contain casts as well. In the old a certain amount of fibrosis of the kidney is not uncommon. A small quantity of albumin present in the urine is not necessarily a sign of renal disease, especially if it is found only occasionally. The albuminuria is often a direct result of the myxœdema and disappears entirely under treatment by thyroid extract, as in one of my own cases.[30]

Generative System.

The external organs of generation may be swollen, the skin covering them dry, and the pubic hair scanty as a part of the general disease. Pregnancy does not usually take place after the onset of myxœdema. This, in many cases, is no doubt because the patients are past the period of child-bearing, but in younger women also it is exceptional, though it does occur occasionally. In the male subject myxœdema does not appear to produce any special change in the sexual power. I know of one case in which the patient married and became the father of two children while suffering from myxœdema.

Amenorrhœa is commonly present in myxœdema, though menorrhagia is not infrequent in the early stages of the disease. The amenorrhœa is present in many cases because the disease has commenced near the natural menopause, but that in some cases it is a symptom of myxœdema is shown by the return of menstruation after treatment by thyroid extract. In one of my own cases, menstruation had occurred only once in the preceding six years, and after treatment with thyroid extract it returned several times, though the woman was over forty-six years of age.

Lymphatic Glands.

The lymphatic glands in various parts of the body are occasionally enlarged. This change is probably an accidental complication only.

Natural Course, Duration, and Termination.

As we now have a means of removing the symptoms of myxœdema and of restoring the patient to health, the disease is not allowed to run its natural course, so that the remarks made under this heading apply only to cases which were observed before the present means of treatment were introduced, when the disease was considered to be incurable, and only palliative treatment was employed. The progress of myxœdema is very slow. The onset is gradual, and it may take many months or even a year or two for the symptoms to become well marked. Occasionally the onset is more rapid. I have seen two cases in which the symptoms were quite distinct, and yet both patients had felt well until six weeks before I first saw them, although the friends of one of these patients had

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noticed that her face was swollen some two months before she herself had felt at all out of health. When once the disease is established there may be but little material change in the general condition for several years at a time, but sooner or later the patient gradually gets worse. Temporary improvement is not uncommon. Most cases improve during the warm summer months, but they relapse into their former condition during the winter. In two cases the symptoms of the disease disappeared during pregnancy but returned again afterwards, one case showed marked improvement for a time after pregnancy.[6] It seems probable, as Hector Mackenzie[31] has suggested, that this temporary recovery during pregnancy takes place because the thyroid gland of the fœtus supplies for the time being the secretion which is deficient in the mother. One case of spontaneous recovery has been recorded by Fraser,[32] of Paisley. In this case the skin was always moist, and profuse perspiration frequently took place. Fraser has been kind enough to inform me that this patient is still alive and has shown no return of the symptoms of myxœdema during the ten years which have elapsed since he recovered. Another such case is mentioned by Hun and Prudden,[7] and a third has been observed by Mrs. Garrett Anderson[33] in which recovery took place without any treatment.

Two cases have been recorded by Köhler in one[16] of which the myxœdema depended upon syphilitic disease of the thyroid gland, and which improved so much after treatment by potassium iodide that the myxœdema was no longer recognizable; in the other[17] the myxœdema was the result of actinomycosis of part of the thyroid gland, and great improvement followed the removal of the infected tissues by surgical means.

As far as I can ascertain these are the only published cases which have terminated in this favorable manner without treatment by thyroid extract, though I am inclined to believe from my own observations that there may be some slight temporary forms of myxœdema which never become well marked, but after a time spontaneously disappear. This is, however, a point which requires further investigation, and is not as yet proved. With these exceptions the disease, in the reported instances, always increased in severity, and sooner or later, unless life was terminated by some intercurrent malady, proved fatal of itself.

The following table, which is compiled from 280 cases, gives the duration of the disease at death or at the time the case was published. As many of the cases were living at the time the record was made, it is evident that these figures give in many instances a shorter period than the full duration of the disease.

Table Showing the Duration of Myxœdema.

Duration.	Males.	Females.	Total.	Duration.	Males.	Females.	Total.
Less than 1 year.....	3	12	15	13 years.....	0	3	3
1 year.....	1	20	21	14 "	0	7	7
2 years.....	4	29	33	15 "	1	7	8
3 "	3	22	25	16 "	0	3	3
4 "	2	24	26	17 "	0	5	5
5 "	4	22	26	18 "	1	1	2
6 "	4	13	17	20 "	0	5	5
7 "	3	11	14	21 "	1	1	2
8 "	4	13	17	24 "	0	1	1
9 "	1	10	11	26 "	0	1	1
10 "	0	17	17	32 "	0	1	1
11 "	0	7	7	34 "	0	1	1
12 "	0	12	12				
				Total.....	32	248	280

The average duration in the 248 women was six years and eleven and a half months, or practically seven years. In the 32 men the average was five years and ten months. We are not, as yet, able to show how long a person with atrophy of the thyroid gland can live by the aid of thyroid extract, but we have no hesitation in saying that life will be much prolonged in these cases by the regular administration of the extract. Considering that my first case of myxœdema, which was cured by thyroid extract, has now continued alive and well for more than four years while taking the extract, we see no reason why loss of function of the thyroid gland should shorten life, provided that the deficiency is made good by constant consumption of the necessary quantity of thyroid extract to maintain health.

Death may occur from a slight attack of pneumonia or bronchitis, or other intercurrent disease to which a patient, enfeebled by myxœdema, offers but little resistance. Death may also take place from cardiac failure or the insanity of myxœdema. We believe, however, that if thyroid extract is systematically used in the treatment of all cases of myxœdema from an early stage very few deaths will result from the disease itself.

Pathological Anatomy.

The Thyroid Gland.—The thyroid gland is found in nearly all cases to be diminished in size, as was first pointed out by Ord [2] in 1877. The gland may be reduced to half its normal size (Buchanan [35]) or to less than a quarter of its usual weight, as in one case examined by Prudden [7] in which it weighed only 112 grains, and in another described by Boyce and Beadles [36] in which the weight was

188 grains, the usual weight of the normal thyroid gland being from one to two ounces (Quain). In exceptional cases the gland may be enlarged, as in a case recorded by Cummins[21] in Thibet. In this case, however, at the post-mortem examination no normal thyroid tissue was found, so that, although enlarged, the gland must have been functionally inactive. On section the lobes are found to be pale and yellowish-white in color, tough and fibrous in consistence. As a rule both lobes are equally affected, but in two or three cases the change has been more marked in one lobe than in the other.

The microscopical examination of sections of the thyroid gland shows in all cases of myxœdema that there is a notable diminution in the amount of glandular structure, and a great increase of fibrous tissue. In the early stages of the disease the walls of the alveoli are seen to be infiltrated by small round cells, while the epithelial cells which line the alveoli generally appear to be proliferating. In the later stages the capsule of the gland is considerably thickened owing to an increase of fibrous tissue in it which is continuous with the stroma of the gland. The fibrous tissue throughout the gland is greatly increased in amount and in certain parts entirely replaces the original gland tissue; it contains large numbers of leucocytes, which are often collected in clumps or gathered round the remnants of the lobules of the gland. The alveoli are much diminished in size and number, and contain little or no colloid material. A few alveoli may still be found lined by epithelial cells, others are represented only by small groups of degenerating epithelial cells embedded in fibrous tissue and surrounded by leucocytes. The walls of the arteries are thickened, especially in the middle and outer coats, and the lumen of the vessel is often considerably diminished or occluded if the vessel is small. These changes are due to endarteritis and hyaline periarteritis as described by R. Boyce and C. F. Beadles.[36] In the most advanced stages the whole gland is practically converted into dense fibrous tissue in which no alveoli remain, though here and there a few degenerated epithelial cells surrounded by leucocytes may still be found. In addition to these changes Prudden[7] has found a newly formed lymphatic tissue surrounding the atrophied lobules and also occurring in separate nodules in two cases of myxœdema. These changes in the structure of the gland are evidently due to a chronic inflammatory process with infiltration of small round cells and formation of fibrous tissue. The process appears to be essentially the same as that which leads to cirrhosis of the liver or kidney. The most important result of the change is the partial or total destruction of the functional activity of the gland. Thus it is evident that, strictly speaking, cirrhosis of the thyroid gland is really the disease

with which we are dealing, and myxœdema is only a symptom or collection of symptoms resulting from this condition, just as ascites is a symptom of cirrhosis of the liver, or general œdema one of renal disease.

The Skin and its Appendages.—The naked-eye appearances of these structures during life have been already described. On microscopical examination the epithelium of the epidermis may be found to be slightly atrophied (Boyce). The connective tissue of the corium shows marked changes, the trabeculæ may be replaced by bundles of fine fibrillæ with nuclei. The lymph vessels are dilated (Prudden). The interfibrillar spaces are unusually wide in some cases so that the fibrillæ and nuclei are seen very clearly. The subcommittee of the Clinical Society, however, concluded that this open-textured condition is exceptional and is similar to that which is produced by ordinary fluid œdema.

The ground substance is considerably increased. Here and there in the upper part of the corium, and especially round the small blood-vessels, there are irregular collections of small round cells. In some cases the blood-vessels show thickening of the walls due to endarteritis, as Ord [2] pointed out in his original paper; in others they appear to be normal. The cutaneous nerves are usually unaltered in structure, though in one case an overgrowth of the perineurium has been observed. In the sweat-glands the epithelium of the coiled tubules is swollen in the earlier stages, then the nuclei proliferate and the lumen of the gland becomes occluded. In the later stages the tubes are found to be surrounded by newly formed fibrous tissue. The sebaceous glands undergo similar changes, and in advanced cases only a few or possibly none of the epithelial cells of the gland may be left. In some cases the changes in the hair follicles are only slight, in others they are extensive. The hair follicles are seen to be surrounded by fibrous tissue with many nuclei, which after a time contracts, causing irregular compression and atrophy of the root-sheath. This leads to the loss of the hair, and in a later stage the follicle may be represented only by a mass of cells occupying its original situation. The same appearances are found in the skin of all parts of the body, though the changes in the hair follicles are naturally seen best where the hair is most abundant in health, as on the scalp. The committee of the Clinical Society considered that these changes in the skin are probably a constant feature in the later stages of the disease.

The morbid condition of the skin is considered by Virchow[44] to be of an irritative character. These alterations in the structure of the skin readily explain the absence of perspiration, the dry skin, and loss

of hair, which are such constant symptoms of advanced myxœdema. Profound though these changes appear to be, it is important to remember that even when the disease has lasted ten or twelve years the skin is capable of restoration to its normal condition, for under the influence of treatment the sweat-glands regain their secretory activity and new hairs are developed from the hair follicles.

Fat.—The increase of weight which so often is a prominent feature in myxœdema is partly due to an increase in the amount of subcutaneous fat, which in nearly all cases is found to be abundant. Fat is also present in considerable quantities, in some cases, in the omentum and beneath the peritoneum. In a few cases it has been described as being œdematous. The fat cells are sometimes smaller than normal, and Boyce and Beadles[36] mention the presence of a vacuole in the nuclei of nearly all the fat cells in one case.

Hypophysis Cerebri.—The condition of the hypophysis cerebri is not mentioned at all in the majority of the reports of post-mortem examinations. In the Clinical Society's report it is stated that no pathological change was found in this gland in the few cases in which it was examined. Since enlargement of the hyphophysis in rabbits, after removal of the thyroid gland, was described by Rogowitsch,[37] Hofmeister,[38] and Stieda,[39] more attention has been paid to this important structure in connection with diseases of the thyroid gland.

In two cases of sporadic cretinism which were examined by Bournevillle and Bricon,[40] and by Dolega[39] distinct enlargement of the hypophysis was found. In two recent papers Boyce and Beadles[36] have described enlargement of the hypophysis in two cases of myxœdema and in one of sporadic cretinism associated with atrophy of the thyroid gland. They also refer to five cases of cretinism recorded by Nièpce[41] in each of which the hypophysis was enlarged. In one case of myxœdema they found that the weight of the hypophysis was doubled. The increase in size was due to hypertrophy of the anterior lobe which, on microscopical examination, showed signs of increased activity as evidenced by an increase in the size of the epithelial cells of the acini and the presence of colloid material both in the acini and in the lymphatic spaces. Somewhat similar changes were observed by Gulland in a case published by John Thomson.[22]

Brain.—Pathological changes in the brain do not appear to be at all constant, as in the majority of cases in which the brain has been examined no change has been found. In one case of myxœdema with insanity, Boyce and Beadles[36] found well-marked endarteritis of the cerebral vessels in both cortex and white matter. There was also considerable periarteritis, causing pressure on the surrounding cere-

bral tissue. They also found colloid material in many of the vessels and perivascular spaces. Similar changes in the blood-vessels of the pia mater and cortex were found by Prudden[7] in a case in which there had been no insanity. Slight hemorrhages, general œdema, increase of fibrous tissue, and cortical atrophy have also been observed in some cases. Whitwell[42] in one case in which there was well-marked delusional insanity found marked vacuolation of the nerve cells and of their nuclei in the gray matter of the motor cortex. This change was most marked in the third and fourth layers of the cortex; there was also distinct increase in the amount of connective tissue throughout the gray matter.

Spinal Cord.—In nearly all cases the spinal cord when examined has been found to be normal. In one case published by Ord[2] in which there was a staggering gait during life, the septa, especially in the posterior columns of the cord, were found to be thickened; in another there was thickening of the pia mater. The anterior horns were degenerated in one case.

Peripheral Nerves.—No change in the structure of the nerves has been described except in one case in which Boyce and Beadles[36] found both perineuritis and endoneuritis of the muscle nerves.

Sympathetic System.—Some change in the superior cervical ganglion appears to be not unusual, as out of ten cases which were examined for the committee of the Clinical Society fibrosis was found in five and probable interstitial change in three.

It will thus be evident that the structural changes which have been described in the nervous system are neither so frequent in occurrence nor so constant in their nature as might be expected from the almost invariable presence of nervous symptoms during life. This probably accounts for the fact that the nervous symptoms, and in some cases even well-marked insanity, disappear under the thyroid treatment, the symptoms in most cases being due rather to a functional than to a structural derangement of the nervous system.

Heart.—The left ventricle is frequently hypertrophied and in association with this there is an increase in the amount of fibrous tissue in the kidneys, the cardiac hypertrophy in these cases probably being the result of the cirrhosis of the kidneys. Increase of fat and fibrous degeneration of the heart muscle are found in some cases. From the frequency with which symptoms of cardiac degeneration occur in advanced cases of myxœdema it is remarkable that so few of the post-mortem records contain any account of fatty or fibrous degeneration of the wall of the heart. On microscopical examination evidences of interstitial myocarditis have been observed in a few cases, notably in one of Ord's.[2] Excess of fat or of fibrous tissue or

of both occurs possibly as a late result of myocarditis. Atheroma and endarteritis of the coronary arteries and their branches are found in some cases, and no doubt tend to bring about degenerative changes in the muscular tissue. Scattered collections of small round cells, lying just beneath the visceral layer of the pericardium in the immediate neighborhood of the blood-vessels, have been described by Prudden;[7] similar cells have been observed by Boyce and Beadles[36] in the same situation.

Arteries.—Atheroma of the larger arteries is frequently found, but rarely in an advanced condition. It was present in more than half the cases examined by the committee of the Clinical Society. Endarteritis of the smaller vessels is of frequent occurrence and may be found in all parts of the body. It not infrequently leads to occlusion of some of the vessels which are affected.

Kidneys.—The kidneys are frequently affected to a greater or less extent by an increase of fibrous tissue. As in other organs there may be also endarteritis. The interstitial nephritis is usually not intense, but may lead to some destruction of the epithelium lining the uriniferous tubules.

Liver.—In the liver, changes occur similar to those already described in the kidney. There is frequently an increase in the amount of fibrous tissue accompanied in some cases by endarteritis and periarteritis.

Lungs.—In nearly all cases only slight or accidental changes have been found in the lungs. In one case Boyce and Beadles[36] describe the lungs as having a gelatinous feel, the walls of the alveoli, the septa, and the pleura being thickened and loose in texture. On microscopical examination there was found to be some small round-celled infiltration of the alveoli, many of which were filled with mucoid substance. The vessels showed marked endarteritis, and in close connection with them there were peculiar "cartilage-like tumors."

Suprarenal Capsules.—Hun and Prudden[7] found collections of small round cells scattered through the gland and around the blood-vessels in one case. In places there was also marked fatty degeneration of the epithelium. Early tubercle was found in one case, otherwise no change has been described.

Submaxillary Gland.—Hale White in one case has described marked increase of the interstitial fibrous tissue, accumulation of epithelial cells in the gland acini and cell-infiltration.

Muscles.—The condition of the muscles has seldom been noted. R. M. Buchanan[35] found that the muscles generally were very pale and soft, and that their fibres were loosely held together. Slight

swelling of the intermuscular connective tissue has been noticed by Boyce and Beadles.[36]

Serous Effusion.—A small amount of anasarca is not uncommonly found. Effusions into the serous cavities are frequently found.

Chemical Pathology.

Skin.—Chemical analyses of the tissues of patients who have died of myxœdema have, in different cases, yielded somewhat varying results. Ord was led to infer from the character of the œdema observed during life and the absence of exudation from the skin when cut after death, that the unusual spaces which he observed between the bundles of fibrous tissue were filled with some gelatinous substance. The committee of the Clinical Society regard these spaces as exceptional, and when present as identical with those seen in ordinary œdema. Cranstoun Charles made an analysis of the skin of the feet of Ord's first patient, who died in the swollen condition, and found that it contained more than fifty times as much mucin as healthy skin or skin swollen by ordinary œdema. It was on account of this mucous nature of the œdema that Ord proposed the name myxœdema for the disease. Subsequent examinations of the skin have failed to show the presence of such a large excess of mucin. Halliburton,[45] in reviewing the results of the analyses of the skin in myxœdema made by Stevenson, Bernays, and himself, points out that in only two was an excess of mucin found; in one 0.81 per cent. and in the other 0.72 per cent. The average of the ten analyses was 0.374 per cent., which, if anything, is slightly less than the normal amount in healthy skin, which has been estimated at 0.385 per cent. Halliburton considers that the amount of mucin found in these analyses was small, in some cases because the patient had died in the atrophic stage of the disease, and in others because the specimens had been kept for a long time in alcohol before the analyses were made. In the swollen stage of the disease there is probably an increase of intercellular ground substance which yields an excess of mucin which is not obtained in the late atrophic stages.

Connective Tissues.—In one case examined by Halliburton[45] the tendo Achillis contained 1.42 per cent. of mucin, which is nearly three times as much as is found in normal tendons. In the cardiac tendons Halliburton has found in each of four cases a slight excess of mucin above the normal amount, and in one case, which had been under the care of Oliver, as much as 5.22 per cent., or five times as much as is usually found in persons who have not suffered from myxœdema. In the same case the muscular substance of the heart

contained 0.26 per cent. of mucin, whereas normal heart muscle contains only minute traces of it. Other organs of the body have been examined for mucin, but the results do not convey much information, as the amount in normal tissues has not as yet been ascertained. The parotid gland, which usually contains merely a trace of mucin, has been examined in only one case, in which the mucin was found to amount to 0.188 per cent., while the submaxillary gland of the same patient yielded 0.159 per cent. (Halliburton). No mucin has been found in either the blood or the urine nor has any been discovered in the pericardial, peritoneal, pleural, or cerebro-spinal fluids.

Pathology.

A consideration of the pathological anatomy of myxœdema has already clearly demonstrated that the one constant and most important change which has been found is the altered condition of the thyroid gland, which is present in all cases. Not only is the gland diseased, but the morbid process is one which gradually leads to destruction of the gland tissue itself to such an extent that the natural functions of the gland can no longer be properly carried on. That this must be so is evident, when we remember that microscopical examination shows that the epithelial cells lining the acini of the gland are gradually destroyed and replaced by fibrous connective tissue. The amount of destruction of gland tissue caused by this process is a most important factor in the production of the morbid phenomena of the disease. The thyroid gland lies in a superficial position and so can be removed without causing injury to the surrounding parts or constitutional disturbance due to the actual operation itself. It will be evident that if the symptoms of myxœdema are really due to a destructive lesion of the thyroid gland, similar symptoms will be caused by simple excision of the gland. This has been proved to be the case by observing the effects of thyroidectomy in various species of animals and in men. The results of thyroidectomy performed experimentally in animals and for disease in men have thrown so much light on the pathology of myxœdema that they require careful consideration.

Results of Removal of the Thyroid Gland in Animals.

In those cold-blooded animals which are able to survive the immediate effects of the operation, extirpation of the thyroid gland appears to produce no effect. Thus Otto Lanz[34] found that removal of the thyroid gland in fifty dogfish (Scyllium catulus and canicula) was

followed by almost entirely negative results, nor was he able to observe any change in twelve lizards (Lacerta viridis) after thyroidectomy. Christiani [83] has found that lizards after thyroidectomy become somnolent and slow in their movements and ultimately die. In adders (*couleuvres*) he has observed a progressive paresis and, in some, casting of the slough and death as a result of thyroidectomy.

In warm-blooded animals two classes of effects follow thyroidectomy. In some animals very acute symptoms come on within a few hours or days of the operation and the animal dies; in others no immediate effect can be observed until some days, weeks, or it may be even months have elapsed, when symptoms appear which closely resemble those of myxœdema in man. In birds the removal of the thyroid gland has not been followed by cachexia strumipriva as has been shown by Allara's experiments on chickens and those of Ewald and Rockwell on pigeons (Horsley [47]). In rodents the earlier experiments tended to show that no results followed thyroidectomy, *e.g.*, in rats and rabbits, but later experiments have shown that there are accessory thyroid glands in this class of animals, and the effects differ according as these are removed with the thyroid gland or left intact. They also show that sufficient time must be allowed to elapse after the operation before definite conclusions can be drawn as to the entire absence of effects due to the thyroidectomy. Schiff and others observed no symptoms after thyroidectomy in rats and rabbits. In rats Christiani [48] found that removal of both thyroid gland and accessory thyroid glands was followed by muscular tremors, dyspnœa, rigidity of the extremities, coma, and emaciation, ending in death in the course of a few hours or days. In rabbits Gley [49] has found that removal of both the thyroid gland and the accessory glands is followed by similar acute cachexia ending fatally within a week, while removal of either the thyroid gland or the accessory glands alone is not followed by any symptoms. This latter conclusion, I believe, may have been arrived at because the animals have not been kept under observation for a sufficiently long period after the operation, for my own experiments, though few in number, show that a condition similar to myxœdema in man can be induced in rabbits by removal of the thyroid gland alone. I have kept three rabbits from which I had removed the thyroid gland alone for a lengthened period. Two of them developed well-marked myxœdema, one eleven and the other twelve months after the operation. The chief symptoms were hebetude, increase in bulk, myxœdematous swelling, dry skin, and loss of hair, all of which are prominent symptoms of myxœdema in man. In ruminants Victor Horsley [6] found in one sheep that removal of the thyroid gland was followed by acute myxœdema, coming on

after an exposure to cold and ending in death 569 days after the operation. The symptoms exhibited were spasms, paralysis, coma, anæsthesia, tetanoid contracture, and subnormal temperature. There was found to be "gelatinous" infiltration of the subcutaneous tissues, which was shown by Halliburton's analysis to be due to mucinous degeneration of the ground substance. Horsley[6] also removed the thyroid gland from one donkey; in this animal also acute cachexia with anorexia, tremor, and fall of temperature came on when the weather was cold, ending fatally 205 days after the operation.

Among carnivora cats have been found to succumb rapidly to acute cachexia following thyroidectomy. In dogs as long ago as 1856–57 Schiff[52] found that the removal of the thyroid gland was followed by a fatal result. In 1884 he again investigated the subject and found that fifty-nine out of sixty dogs succumbed after total extirpation of the thyroid gland. Other observers have obtained similar results. These animals in the majority of cases succumb to the effects of thyroidectomy at the end of a period which varies from a few days to four or five weeks, rarely extending to two or three months. Anorexia and hebetude are early symptoms. Fibrillar muscular twitchings soon appear and the animal has attacks of clonic convulsions which affect all the trunk and limb muscles, accompanied by marked dyspnœa. There is great weakness of the limb muscles and rapid emaciation. The temperature rises at first and then falls below the normal level. The red corpuscles are diminished while the white corpuscles become more numerous. The animal becomes comatose and finally dies. O. Lanz[34] has shown that the origin of the muscular twitchings in the cachexia thyreopriva of dogs lies in the medulla oblongata, though the tetanic convulsions are under the influence of the higher nerve centres as well. He also draws attention to the fact that other symptoms which occur, such as vomiting and difficulty in swallowing, are also of bulbar origin.

In monkeys the results of thyroidectomy have been investigated by Munk and by Horsley. The results which were obtained by Victor Horsley[51] have a most important bearing upon myxœdema in man. In monkeys the symptoms first appear about the fifth day after the operation. The length of the latent period depends upon the age of the animal and the temperature of the atmosphere in which it has been kept, being shorter if the animal is young or if it is exposed to a low temperature. The writer has removed the thyroid gland from four bonnet monkeys and from one rhesus monkey. All the bonnet monkeys, which were kept at a temperature of from 60° to 70° F., developed acute myxœdema. The rhesus monkey remained

well for nearly fifteen months. When it was killed the immunity from myxœdema was explained by the presence of a piece of thyroid gland tissue, which was apparently either a portion of the gland left at the operation or an accessory gland which had hypertrophied sufficiently to maintain the health of the animal.

The symptoms of acute experimental myxœdema last on the average between three and four weeks. The earlier symptoms are almost entirely nervous in origin. Fine tremor in the limbs, clonic spasms, and contracture of the limbs are usually present, accompanied by a general diminution of voluntary power. Extensive "functional" paralysis may occur, and Horsley [6] has observed in two cases complete hemiplegia which passed off after a few hours. One of the monkeys from which I removed the thyroid gland was twice observed to fall suddenly off his perch, the fall being followed by a period of unconsciousness accompanied by tonic muscular spasm. These attacks, from which recovery took place after a few hours, appeared to be epileptic. Another monkey, shortly before death from acute myxœdema, had several epileptic convulsions characterized by tonic muscular contractions with risus sardonicus, followed by clonic contractions and micturition. In one there was convergent strabismus with retraction of the eyelids, and in another turning of the head and eyes to the left with lateral nystagmus. The sensory symptoms are paræsthesia with anæsthesia later. Mental dulness and apathy are well marked, and coma may finally close the scene. The temperature is first raised and irregular, undergoing considerable daily variations; it then becomes subnormal, and I have found it as low as 80° F. in the rectum four days before death. The appetite is increased at first, but diminishes afterwards. Well-marked changes take place in the blood, the red-blood corpuscles being diminished in number while the leucocytes become more numerous. The face becomes swollen with gelatinous œdema, especially marked in the eyelids; the skin is dry and rough, and the hair may fall out just as in myxœdema in man. The amount of mucus secreted by the intestinal canal and bladder is increased, and the parotid gland has been found to secrete mucus also. Mucinous degeneration of the connective tissues and wasting also take place. Horsley [6] has shown that heat has a remarkable influence upon the course of the symptoms in experimental myxœdema. He found that by keeping the animals at a temperature of 90° F. the average duration of life after the operation was increased to 125 days instead of 24 days, the average duration at ordinary temperatures, and that the symptoms were considerably modified. In this chronic form of experimental myxœdema the symptoms closely resemble those of cretinism in man.

Chemical Pathology.—The tissues of several species of animals have been examined chemically by Halliburton[45] after the thyroid gland has been removed. In a pig and in a donkey no excess of mucin was found. In the case of a sheep which developed myxœdema two years after thyroidectomy, the urine contained an abundance of mucin; the connective tissue of the anterior angle of the neck contained 0.9 per cent. of mucin, which Halliburton considers an indication of a considerable increase in the amount of mucin. The most positive results have been obtained in monkeys which, as we have already seen, develop a typical form of myxœdema after thyroidectomy. In these animals the percentage of mucin in the tissues is notably increased after the operation. A fact of special interest is the increase of mucin in the salivary glands and especially the presence of mucin in the parotid gland, which in the healthy animal does not contain any at all. Mucin is also found in the blood in increasing quantities as the myxœdematous condition develops, so that in the monkey we have a true mucous œdema. In some cases the blood clots very slowly, and a well-marked buffy coat is formed. In one monkey which was kept at a high temperature and which was in consequence not myxœdematous, no excess of mucin was found.

That the effects described are entirely due to the removal of the thyroid gland alone is further shown by the fact that other injuries in the same region do not produce these symptoms. Thus Schiff[52] found that exposure of the thyroid gland or section of its nerves did not cause any of the symptoms which followed total extirpation of the gland. Munk[53] and Drobnick[54] have each attributed the symptoms of cachexia thyreopriva to irritation of the nerves in the neck caused by the operation, but this theory has been proved to be untenable by Schiff's experiments and also by those of Fuhr, Herzen, and Fano. Fuhr[55] has shown that no injury of the nerves in the thyroid region is capable of producing the same results as thyroidectomy. Herzen[56] found that injuries inflicted on the muscles, vessels, and nerves of the neck did not cause cachexia thyreopriva. Fano and Zanda found that if the gland was ligatured but left *in situ*, the same symptoms occurred as when it was removed, because by this operation the gland was rendered functionless. If one lobe of the gland was removed and the other dislocated, the vessels and nerves being left intact, no symptoms followed; but if this lobe was also removed, then the usual symptoms appeared. Further evidence of the same nature is afforded by experiments in which only partial thyroidectomy has been performed. Thus Colzi, Fuhr, and Rogowitsch have shown that as long as part of the thyroid gland is left to

carry on the necessary function, no symptoms appear. In many cases half of one lobe or even one-third of a lobe, was found by Sanquirico and Canalis [57] to be sufficient for this purpose. In some cases the lobe or portion of a lobe which is left after a partial thyroidectomy undergoes hypertophy, in others it does not. This is a point which requires fuller investigation. These facts will be found to hold good in the case of man in whom the evil effects of total thyroidectomy can be avoided by leaving a part of the gland at the time of the operation, a procedure which is nearly always adopted at the present day. A consideration of these facts shows that in certain of the lower animals, and especially in monkeys, a condition of myxœdema identical with myxœdema in man can be induced by simple ablation of the thyroid gland. Thus experimental pathology, like morbid anatomy, proves that the symptoms of myxœdema are due to loss of function of the thyroid gland.

Results of Removal of the Thyroid Gland in Man.

The effects which have been produced in man by total extirpation of the thyroid gland for goitre have thrown much light upon the pathology of myxœdema. On September 13th, 1882, at a meeting of the Medical Society of Geneva, J. L. Reverdin [4] drew attention to certain changes which he had observed to take place in some of his cases after total extirpation of the goitrous thyroid gland. The chief symptoms of this condition were weakness, pallor, anæmia, and œdema, without albuminuria, and he mentions that one patient closely resembled a cretin in appearance. On April 4th, 1883, at the twelfth congress of German surgeons, Kocher [5] gave a very complete account of the symptoms which he had observed, with but few exceptions, to follow total removal of the thyroid gland for goitre, and to which he gave the name "cachexia strumipriva." This condition was considered by Kocher to be the result of injuries sustained by the tissues in the neck during the operation, rather than the effect of the loss of the thyroid gland. Soon afterwards a further communication was published by J. L. and A. Reverdin [58] in which attention was directed to the similarity between this condition following total thyroidectomy and myxœdema, which led them to name it "myxœdème post-opératoire." These authors pointed out that this peculiar condition was developed only after total extirpation of the thyroid gland, and rightly attributed it to the loss of that gland, a view which was afterwards adopted also by Kocher. The symptoms of the cachexia frequently begin to appear soon after the patient has recovered from the immediate effects of the operation; in some cases, however, no change has

been observed until four or five months have elapsed. In many cases the earliest symptoms are pains in the limbs and sometimes also in the shoulders, neck, and abdomen; these are soon followed by languor with weakness of the limbs, which, in the absence of early pains, may be the first symptoms to appear. Coldness of the hands and feet is complained of, especially in cold weather. Slowness of mental processes soon becomes a prominent feature, the speech is slow, and all voluntary movements are executed much more deliberately than before the operation. The sufferers are generally perfectly aware of the loss of their former activity. Swelling of the face, hands, and feet then takes place. The swelling may come and go for a time and then become permanent. It is most conspicuous in the eyelids, and may cause pendulous swellings of the lower eyelids. As a result of the general swelling the features are much altered, the nose and lips become large and thick, and the whole face is broadened. The trunk also is increased in circumference by the swelling. The skin becomes dry, the superficial layers of the epidermis may peel off in places, and the hair also may be lost. The anæmia often becomes marked. Some of the less common symptoms observed by Kocher were dysphagia, giddiness, retching, fainting fits, spasms, epileptic attacks, etc.

From this description it will be seen that the clinical resemblance between cachexia strumipriva and idiopathic myxœdema is complete. So striking is the similarity in appearance between the two that Otto Lanz,[34] who had often examined Kocher's cases of cachexia strumipriva, on seeing a photograph of one of the writer's cases of myxœdema in Newcastle, thought that it was taken from one of these cases which he had himself photographed in Berne! Kocher[5] found that of thirty-two patients upon whom he had performed total thyroidectomy for goitre, twenty-six developed the cachexia. The other six remained free from the cachexia, but in every one of them a return of the goitre developed either from a remnant of thyroid tissue not removed at the time of the operation or from an accessory thyroid gland. Thus in those cases in which sufficient thyroid gland tissue had been left no cachexia appeared, while the symptoms which developed in the others were evidently due to complete absence of the gland. The facts collected by the committee of the Clinical Society from a number of different surgeons showed that cachexia strumipriva had developed in about one-third of the cases in which apparently all active thyroid tissue had been removed, and in only a few cases after partial thyroidectomy. This evidence does not prove that the cachexia is due to the loss of thyroid tissue alone. When, however, we consider that these symptoms never follow any other operation

involving the other important structures in the neck, it is evident that the cachexia when it develops is the result of removal of the thyroid tissue. Why it does not develop in all cases after total thyroidectomy we are not in a position to say. It seems likely that in these cases a portion of the gland tissue which has been left unobserved at the time of the operation, of an accessory thyroid gland as described by Sandström,[59] or even of some other gland, has undergone sufficient compensatory hypertrophy to enable it to carry on the functions of the gland which has been removed, and so prevent the onset of the cachexia.

Restoration of Thyroid Function by Transplantation of Healthy Gland Tissue.

Schiff was the first to show that the fatal effects of thyroidectomy in dogs could be averted by transplanting the thyroid gland of another dog into the abdomen of the animal before the thyroid gland is removed. Von Eiselsberg,[60] by a series of experiments on cats, found that if one lobe of the thyroid gland was removed and transplanted into the subperitoneal tissue or into a fold of mesentery, and the graft was successful the animal did not succumb after subsequent removal of the other lobe of the gland from the neck. In two animals in which this was successfully accomplished, post-mortem examination showed that the transplanted gland had become vascular and so had been enabled to continue its original functions in a new situation. In the animals which had succumbed to the usual symptoms, it was found that the graft had failed. Victor Horsley,[9] who first drew attention in England to these experiments, pointed out that they afforded conclusive proof that if a grafted thyroid gland lived after transplantation, it could carry on its functions so as to avert the usual results of thyroidectomy. From this important conclusion Horsley was led to suggest that the inevitable progress of myxœdema, cretinism, and cachexia strumipriva to a fatal termination might be arrested by grafting a piece of healthy thyroid gland beneath the skin of the patient so as to restore the lost function. In a later communication Horsley[62] states that in 1883 Kocher had already tried transplantation of a piece of freshly excised goitre, but without success, and that Bircher[63] in 1889 transplanted a piece of apparently normal thyroid tissue from a goitre into the abdominal cavity of a patient suffering from cachexia strumipriva. Great improvement followed for a time, and then a relapse occurred. Still greater improvement followed a second transplantation.

This subject will be considered more fully in connection with the

treatment of myxœdema; all we wish to indicate here is that the results of successful thyroid grafting have proved that in man as well as in animals the functions of the gland can be carried on after thyroidectomy by a piece of transplanted thyroid tissue as long as it lives, but that if it atrophies the symptoms of cachexia thyreopriva appear. We can, however, now go a step further and show that the thyroid gland exercises its remarkable influence upon the general metabolism of the body by means of a secretion which is formed by the epithelial cells of the alveoli of the gland.

King, Baber, Boéchat, and Horsley [64] all found that the colloid material which collects in the acini of the gland escapes into the lymphatics and thus passes into the general circulation. Hürthle [65] states that ten days after exision of five-sixths of the thyroid gland, microscopical examination of the remaining portion revealed evidences of increased secretory activity. Wyss has also shown that the injection of pilocarpine causes increased secretion of the colloid substance. Hürthle has found that the colloid material may escape into the lymph spaces either by rupture of the wall of the acinus or by passing along minute channels between the epithelial cells. There is thus ample evidence from microscopical appearances presented by the gland that it is a true secretory gland, the secretion from which, instead of passing along a duct as does that of such glands as the salivary, finds its way by the lymphatics directly into the general blood stream. We have, however, experimental evidence showing that this secretion, when separated from the gland, is still capable of carrying on the functions of the thyroid in the absence of the gland itself.

In 1890 Bettencourt and Serrano [10] reported a case of thyroid grafting in a woman suffering from myxœdema in which great improvement followed the operation. The improvement, however, commenced as early as the day following the operation. The authors, therefore, came to the conclusion that it could not be due to a complete vascularization of the gland, but rather to an absorption of the juice of the grafted gland by the tissues of the patient. On reading the report of the case the writer was struck by the great importance of this observation. Taken in conjunction with many of the facts bearing on the functions of the thyroid gland which we have already considered, this observation appeared to him to afford the strongest evidence of the physiological activity of the secretion of the gland. This appeared easy to test, for if the function of the thyroid were really secretory the administration of the secretion alone to those in whom the gland had become functionless through disease or operation would be followed by a great improvement in

their condition; just as the administration of pepsin and hydrochloric acid improves the digestion when the gastric secretion is deficient in these substances. The writer, therefore, determined to test the action of thyroid juice, given in the form of a glycerin extract of sheep's thyroid glands, in a case of myxœdema as soon as the opportunity presented itself. G. Vessale,[66] directly after total thyroidectomy in nine dogs, gave intravenous injections of an extract made from the thyroid glands which he had just removed, in order to prevent the development of cachexia strumipriva. He found that the venous blood which had become very dark became normal again in a few hours, and the "neuroparalytic hyperæmia" of the conjunctiva diminished, illustrating the beneficial effect of the injections. Soon afterward, on April 13th, 1891, the writer[12] tested the action of a glycerin extract of a sheep's thyroid gland in a well-marked case of myxœdema. The extract was injected beneath the skin of the patient, so that it might be slowly absorbed by the lymphatics and so enter the circulation in a manner as closely as possible resembling that in which the secretion of the gland enters the blood stream in a healthy person. The author was able to show that by repeated injections the symptoms of myxœdema can be entirely removed, and that the function of the thyroid gland can be carried on by the secretion alone in the absence of the gland itself.

The results of injections of thyroid extract in animals after thyroidectomy are equally instructive. In these experiments the symptoms are brought about by a sudden cutting off of the supply of the thyroid secretion by removal of the thyroid gland. They thus afford a more stringent test of the secretory function of the thyroid gland than cases of myxœdema in man in which the symptoms are due to a gradual and perhaps only a partial loss of the thyroid secretion. Gley has found that in the case of dogs and rabbits the usual effects of thyroidectomy may be averted or considerably lessened by injections of thyroid extract. In a monkey the symptoms of acute myxœdema induced by thyroidectomy were removed by the writer[68] by means of repeated injections of thyroid extract. In this experiment the whole thyroid gland was removed from a bonnet monkey, which was kept in a room the temperature of which was between 62° and 66° F. At the beginning of the second week after the operation a fine tremor of the upper limbs appeared. During the next few days the monkey became much less active than usual, the tremors became more marked, and there were occasional clonic contractions of the muscles of the arm and forearm. A slight myxœdematous swelling of the upper and lower eyelids appeared; the symptoms increased till in the fourth week the monkey was very inactive, sitting still

nearly all day with the trunk bent forward. The tremors were well marked, the myxœdematous swelling of the face was very distinct. The temperature chart showed considerable diurnal variations, the morning temperature being subnormal on three occasions. The red blood corpuscles had diminished to three-fourths of their original number. When this stage had been reached thyroid extract was injected beneath the skin in doses of one or two minims. The first injection was made on the twenty-sixth day after the operation, and between this and the forty-fourth day fourteen injections were made. The total amount of extract employed was only twenty minims, only one-third of which was really active, as two-thirds of the extract consisted of a mixture of equal parts of glycerin and very dilute carbolic acid. As a result of the injections the monkey steadily improved. The tremor vanished, the myxœdematous swelling disappeared, the temperature, which soon after the commencement of the injections had fallen as low as 95.4° F., became normal, and the red blood corpuscles increased up to their normal number. The animal regained his former activity so that, with the exception of his weight being rather less than before the operation, he was quite well again by the end of the seventh week. The injections were discontinued and the symptoms returned, the animal ultimately dying of acute myxœdema and dysentery.

The total absence of the thyroid gland was proved by post-mortem examination. The recovery of this animal from the symptoms of experimental myxœdema shows that the loss of the thyroid gland was compensated for by the use of the thyroid extract as long as the injections were continued. It is thus evident that the thyroid is a secretory gland supplying what has been aptly termed by Brown-Séquard "an internal secretion."

The pathology of myxœdema may be summed up shortly thus:

1. The usual change found in cases of myxœdema is a fibrosis of the thyroid gland leading to a destruction of the alveoli of the gland and of the epithelium which lines them—a change which must necessarily diminish, if it does not entirely suspend, the functional activity of the gland.

2. A condition of myxœdema can be induced in certain of the lower animals, especially in monkeys, by removal of the thyroid gland.

3. Total extirpation of the thyroid gland in man has been followed in a considerable number of cases by a myxœdema, identical with idiopathic myxœdema, which could be attributed to no other cause than the loss of the thyroid gland.

4. In animals the usual results of thyroidectomy can, to a large extent, be obviated by successful thyroid grafting.

5. In man the symptoms of myxœdema have been greatly improved, and in some cases entirely removed, by grafting pieces of healthy thyroid gland, the early improvement being due to absorption of the juice of the gland.

6. In some lower animals the acute symptoms following thyroidectomy can be removed by injections of thyroid extract, and in the monkey the symptoms of experimental myxœdema, induced by previous thyroidectomy, can be entirely removed by the same means.

7. The symptoms of myxœdema in man can be entirely removed by the administration of thyroid extract, either by hypodermic injections or by the mouth.

These facts conclusively prove that all forms of myxœdema are really varieties of one and the same disease, and that all are due to loss of function of the thyroid gland, that function being to supply an internal secretion which is essential to the proper maintenance of the health of mind and body.

Diagnosis.

The diagnosis of a well-marked case of myxœdema is readily made. The appearance of the patient is so peculiar that the nature of the case can be recognized at a glance. The solid œdema producing the characteristic appearance of the face, the condition of the skin and hair, the hebetude, the slowness of thought, speech, and action, and the subnormal temperature, combine to form a clinical picture which is quite characteristic.

The solid œdema has sometimes been mistaken for the œdema of renal disease. An examination of the urine, however, at once reveals the fact that the condition is not dependent upon disease of the kidney. The onset of myxœdema is gradual, and the diagnosis must be made as soon as possible in the early stages of the disease long before it has become fully developed. Under these circumstances slight evidences of the disease may be readily overlooked and the symptoms on the part of the nervous system, which may occur during the early stages, may be attributed to some other cause. Slight degrees of myxœdema are by no means rare in women between forty and fifty years of age. In these cases the patient complains of chilliness, of some loss of memory, of languor, and possibly of other ill-defined subjective symptoms. Not uncommonly the symptoms are attributed to the menopause. In these cases a careful examination of the patient may reveal the presence of some brawny swelling of the cheeks with swelling of the eyelids at times. In some the myxœdematous swelling may closely resemble ordinary obesity. The speech

also is often slightly drawling in character. If a doubt arises as to the true nature of these symptoms, a simple test is to treat the patient for a time with small doses of thyroid extract. If the symptoms are due to changes in the thyroid gland marked improvement will take place in a short time. The swelling diminishes and the subjective symptoms disappear; such improvement taking place after the administration of thyroid extract at once confirms the diagnosis of thyroid disease.

Once the diagnosis of myxœdema has been made, the next step will be to determine the nature of the disease which has led to the diminution of the functional activity of the gland. In the great majority of cases the myxœdema is due to chronic interstitial thyroiditis, which is characterized by a gradual onset, and by the diminution in the size of the thyroid gland, due to the contraction of the newly formed fibrous tissue in it. The gland then becomes so small that it can no longer be detected on palpation of the neck. If there is a history of a previous operation upon the thyroid gland or in its immediate neighborhood, the symptoms are most probably due to removal of too large a portion of the gland at the time of the operation. In a few cases the thyroid gland will be found to be enlarged; in these cases goitrous degeneration has led to the destruction of the gland tissue proper, or possibly the gland tissue has been destroyed by a new growth. It must also be remembered that in rare cases syphilis or actinomycosis of the thyroid gland may be the cause of the symptoms.

Prognosis.

The prognosis of myxœdema has entirely changed since the introduction of the present methods of treatment. Formerly a certain amount of improvement might be expected in warm weather, and from the employment of diaphoretics, especially pilocarpine, massage, warm baths, and general tonics, but sooner or later the patient was sure to get worse and die of the disease. Under the regular administration of thyroid extract the symptoms of myxœdema can now be entirely removed in a few months, and by the continued administration of the remedy any return of the symptoms can be prevented. This freedom from myxœdema can be maintained apparently as long as the patient lives. From this it follows that if treatment is commenced early and there are no complications, life should not be appreciably shortened by the changes in the thyroid gland. If complications are present, or if the case is one of long duration, the prognosis is not so favorable and must be made according to the condition of the patient at the commencement of thyroid treatment.

The presence of advanced cardiac or arterial degeneration is always a source of danger, and thyroid treatment has, in such cases, been followed by a fatal result. Insanity increases the gravity of the prognosis, but it is not necessarily incurable, for several cases have recovered from the insanity of myxœdema under the new treatment. If albumin is present in the urine, but disappears after treatment, its presence does not materially affect the prognosis. If, however, albuminuria persists and there are casts in the urine, the prognosis will be governed by the nature and gravity of the disease of the kidney. If any other complications occur their bearing upon the future welfare of the patient must be estimated according to their character and severity.

Treatment.

The great improvement in our methods of treating myxœdema which has taken place during the last few years affords one of the most striking illustrations of the benefits which practical therapeutics may derive from the application of knowledge gained in the field of experimental pathology. As recently as the year 1888 myxœdema was considered to be a hopelessly incurable disease, and the most that could be done was to endeavor to relieve the more distressing symptoms as they arose. Tonics such as the hypophosphites, quinine, and iron were employed to relieve the general debility which is so often a prominent symptom; the beneficial effects were, however, only temporary. Nitroglycerin in a few cases was found to be of service. Jaborandi and pilocarpine were frequently employed and often afforded considerable relief, helping to moisten the dry skin. It is also probable that some of the beneficial effects observed after the use of pilocarpine were due to an increased activity in small portions of glandular tissue which still remained in the diseased thyroid gland. As might be expected from the low temperature which is found in myxœdema, warmth has been found to be of considerable service. The removal of the patient to a warm climate, such as that of Egypt during the winter, not only relieved the sensation of cold, but tended to prolong life. With the introduction of thyroid grafting for myxœdema we come to a most important advance in the direction of the rational treatment of the disease.

Thyroid Grafting.

We have already considered in the section devoted to pathology the various clinical and pathological facts which have clearly pointed out that myxœdema occurs as a result of loss of the function of

the thyroid gland. When once this important fact became firmly established the practical application of it to treatment resulted in attempts to restore the lost function, for it had become apparent that if this could be accomplished the progress of the disease would be arrested. In order to accomplish this, attempts were made to graft pieces of thyroid gland into various parts of the patient in the hope that they might become sufficiently vascularized to retain their vital activity and so be able to carry on the functions of the original gland, which had either become useless or had been removed. As early as 1883 Kocher grafted a piece of a goitre which had just been removed; this, however, was absorbed. In January, 1889, Bircher [63] grafted a piece of what appeared to be healthy thyroid tissue into the abdominal cavity of a patient who had developed myxœdema after removal of the thyroid gland. As a result of this operation the patient recovered sufficiently to return to work, and lost nearly all the symptoms of myxœdema. Three months afterwards, owing to atrophy of the transplanted piece of thyroid tissue, the symptoms of myxœdema returned. A second operation was followed by even greater improvement, which lasted for nine months. This case thus firmly established the value of this method of treatment if the operation was successful, and showed that it was sufficient as long as the graft did not atrophy.

Previous to the publication of this case in 1890, Victor Horsley, [62] as a result of the successful thyroid grafting performed by von Eiselsberg [60] on cats, had already suggested in England the transplantation of a healthy thyroid gland from one of the lower animals into the peritoneal cavity or into the subcutaneous tissue "as a possible means of arresting the progress of myxœdema, cachexia strumipriva, and allied diseases." Horsley also recommended the employment of the thyroid gland of the sheep, for it resembles that of man in structure, and the symptoms which follow thyroidectomy in the sheep resemble those which follow the same operation in man. As a result of the success of Bircher's case, Kocher has again several times tried transplantation of the fresh thyroid gland of both man and lower animals into the abdomen; in each case only a temporary improvement followed. Kocher and Lanz [34] have tried the implantation of the thyroid gland of the dog by fixation in the tunica vaginalis, and also in the femoral artery. In one case there was a slight temporary improvement; in the other none at all. Bettencourt and Serrano [10] introduced one-half of the thyroid gland of a sheep into the subcutaneous tissue of the inframammary region on each side of a woman of thirty-six who had suffered from myxœdema for several years. The operation was followed by an immediate improvement, the first indi-

cation of which was an elevation of the previously subnormal temperature. The number of red blood corpuscles increased within a month from 2,442,000 to 4,447,000 in the cubic millimetre. Movements became more easy of execution and the speech clearer. Perspiration, which had entirely disappeared, again returned. The swelling of the body decreased and the weight diminished from 119 kil. ½ to 113 kil. 800. The menstrual period which followed the operation lasted only four days, whereas previously it had lasted two and sometimes three weeks. As the improvement commenced the day after the operation, the authors rightly concluded that it was the result of the absorption of the juice of the thyroid gland by the tissues of the patient and could not be due to the grafted glands becoming vascularized, and so functionally active. In the majority of cases in which this operation has been performed, the improvement has been temporary, showing that the grafted gland has not become sufficiently vascularized to carry on its function permanently. MacPherson [69] has treated a case by this method in which such prolonged improvement has resulted that it is evident that the grafted gland tissue has become vascularized. In this patient, a woman of thirty-nine, with well-marked myxœdema and melancholia, the operation was performed in the same manner as in Bettencourt and Serrano's case. Within twelve hours there was a marked improvement in the mental condition. In a short time the temperature ceased to be subnormal, the nervous and mental symptoms and the headache disappeared; the anæmia was relieved, and the skin became soft and smooth; menstruation became regular. It is now nearly three years since the operation was performed, and MacPherson has been kind enough to inform me that the patient is now quite well. She has had two attacks of mental disturbance of short duration, but there has been no return of the myxœdema.

More recently Victor Horsley [70] has suggested that it is advisable to remove the symptoms of myxœdema before the operation by the administration of thyroid extract so that the grafted gland may be embedded in normal instead of diseased connective tissue—a suggestion which, as far as I am aware, has not yet been put into practice. Thyroid grafting, if successful, is a most satisfactory method of treating myxœdema. As yet the grafted gland has so often failed to become vascularized that since the introduction of the treatment by thyroid extract this method has seldom been utilized. We do not yet know what is the cause of the chronic interstitial thyroiditis which is the essential factor in nearly all cases of myxœdema. It may be that the cause of the original change in the thyroid gland still remains in operation and either prevents the

grafted gland from developing or else leads to its early destruction. If this be the explanation of the failure of the operation in some cases of myxœdema, it would not, of course, account for its want of success in cases of cachexia strumipriva. When this method of treatment is employed certain precautions are necessary in carrying it out. For two or three months before the operation a systematic course of treatment by thyroid extract should be carried out, as suggested by Horsley,[70] in the manner described in the next section. This treatment must be continued until the symptoms of myxœdema have disappeared and the skin and subcutaneous tissues have returned to their normal condition. The thyroid gland which is to be used for the graft should be selected from a perfectly healthy sheep which is fully grown, but not more than a year old. The sheep should, if possible, be slaughtered near to the operating theatre and only when the patient is ready for the operation. It should be killed by a blow or a shot through the head with a bullet rather than in the usual manner by bleeding. A median longitudinal incision is made in the neck of the sheep, one lobe of the thyroid gland is then dissected out rapidly with a sterilized knife and forceps and transferred to a warm sterilized glass jar in which it is taken at once to the operator. An incision is made through the skin and subcutaneous fat beneath each breast of the patient, the lobe of the thyroid gland of the sheep is slit in two halves, and one half is placed in the subcutaneous tissues on each side. The wounds are closed and dressed with the usual aseptic precautions. It is advisable, however, that no chemical antiseptic should be introduced into the wound itself, but that sterilized water be used instead for cleansing it. If the symptoms of myxœdema reappear within three or four months it is an indication that the graft has failed; if, however, six months pass without any return of the myxœdema it will be evident that the gland tissue has become vascularized and is furnishing a sufficient supply of its secretion to keep the patient in health. If the original graft fails, either at once or after it has been functionating for a time, as shown by the return of the myxœdema, the operation may be repeated. It is probable, however, that the great majority of patients will prefer treatment by thyroid extract, which is very simple though it has to be continued permanently.

Treatment by Thyroid Extract.

Theory and Development of the Treatment.—We have already considered the various observations which, having showed that myxœdema was due to loss of function of the thyroid gland, led to the

suggestion of thyroid grafting as a means of treatment. It will be remembered that the adoption of this suggestion by Bettencourt and Serrano was followed by marked immediate improvement in the condition of their patient, commencing the day after the operation. This early improvement was attributed by these authors to the absorption of the juice of the grafted thyroid gland by the tissues of the patient. This observation was reported to the Association Française pour l'Avancement des Sciences, on August 9th, 1890.[10] On reading the report of this case the writer was struck by the importance of the suggestion that such a marked improvement in a case of myxœdema might be due to the simple absorption of thyroid juice, for it appeared to him that if this were the correct explanation of the improvement the same result could be obtained by the employment of the thyroid juice by itself as a remedy for myxœdema. By supplying the patient with repeated doses of the secretion of the gland it was hoped to maintain a greatly improved state of health indefinitely. A few months afterwards the opportunity of testing this method of treatment presented itself in the shape of a patient with well-marked myxœdema who was perfectly willing to allow the efficiency of the remedy to be tested in her case. Before the treatment was tried the patient was shown on February 12th, 1891, at a meeting of the Northumberland and Durham Medical Society, on which occasion the employment of thyroid extract as a remedy for myxœdema was first publicly suggested by the writer[11] in the following words: "The symptoms, then, are due to the absence from the system of some substance which is secreted by the normal thyroid gland. . . . It has occurred to me that it would be worth while to try the hypodermic injection of an emulsion or extract of the thyroid gland of a sheep. This treatment appears to be rational, and, at any rate, would not harm the patient. We may, I think by this means, hope to produce as much improvement in a case of myxœdema as was brought about by the absorption of the juice of a piece of the gland which had been introduced beneath the skin, and it has the advantage of being a much simpler proceeding."

The most suitable form in which to employ the remedy was found to be a glycerin extract of the thyroid gland of a sheep diluted by a weak solution of carbolic acid. As the thyroid secretion in health passes by the lymphatics from the gland into the blood, it was determined to inject the extract beneath the skin so that it might be slowly absorbed by the lymphatics and carried into the blood stream in a similar manner. It was evident that the efficiency of the remedy would be fairly tested by this method of administration. The thyroid gland of the sheep, which had been recommended for grafting by

Horsley and had been proved to be suitable for this purpose by Bettencourt and Serrano, was selected for the preparation of the extract. Sheep are generally healthy and rarely suffer from tuberculosis. They are killed in large numbers every week in all towns so that a supply of fresh thyroid glands can easily be obtained when required. The extract is prepared in the following manner: As soon as the sheep has been killed and the skin has been removed from the neck, a median incision is made down to the larynx and trachea. The muscles on each side are then held apart and a little dissection with sterilized instruments soon exposes the two lobes of the thyroid gland, lying one on each side of the larynx and upper part of the trachea. The lobes are dark reddish-brown in color, firm in consistence, shaped like an almond and united by a rudimentary isthmus crossing the front of the trachea, which may be easily overlooked. Each lobe is removed separately and transferred to a sterilized glass jar. When the sheep is opened the thoracic and abdominal organs must be inspected to make sure that the animal is healthy. If there are any signs of disease in the animal or in the gland itself it must be discarded. The thyroid gland of a pig or a cow may be used if it is more easily obtained. All the apparatus employed in the actual preparation of the extract should be previously sterilized either by dry heat or by boiling. The glands are freed from any fat or connective tissue which may remain adherent to them, they are then finely minced and the fragments, together with the fluid which has escaped during the process, are placed in a mixture of equal parts of glycerin and a half-per-cent. solution of carbolic acid or of glycerin and boiled distilled water, in the proportion of two cubic centimetres of the mixture to each thyroid lobe. The carbolic acid is not really necessary but was employed in the earlier preparations. The mixture is allowed to stand in a cool place for from twelve to twenty-four hours, it is then squeezed through a fine cloth by means of a press so as to obtain as much liquid as possible. By this means three cubic centimetres or forty-five minims of the liquid thyroid extract are obtained from each lobe of the gland, that is to say, on the average six cubic centimetres or one drachm and a half from the entire gland. Thyroid extract was first used in the treatment of myxœdema by the writer[12] on April 13th, 1891.

The patient to whom reference has already been made was a married woman, aged forty-six, who had suffered from myxœdema for four or five years, and presented all the chief symptoms of the disease. The injections of thyroid extract were made with antiseptic precautions, and in the course of six or seven weeks the improvement in the condition of the patient was so marked that the efficiency of

the remedy could no longer be doubted. During the first three months the extract of two and a half thyroid glands was injected usually in doses of twenty-five minims at a time. At the end of this period the myxœdematous swelling was greatly diminished and had almost entirely disappeared in some situations. This diminution in swelling was very distinct in the face. The speech was more rapid and fluent, the mind more active, and the memory more retentive. The sense of languor was greatly diminished while the bodily activity was increased. Menstruation returned after amenorrhœa of four years' duration. The skin became soft and moist and perspiration, which had not been seen for four years, reappeared after exercise. The temperature became normal. This remarkable improvement clearly proved the efficiency of the treatment and the correctness of the theory upon which it was founded. This result was communicated to the British Medical Association at its annual meeting in July, 1891.[12] Four months after the suggestion of this method of treatment had been published by the writer[11] and two months after he had actually commenced to employ thyroid extract, several trials of the same method were made in France. In June, 1891, Gley and Magnan[71] treated two cases of myxœdema in Paris by injections of thyroid extract. Two injections were given in one case and four in the other. During the same month Merklen[72] also made a trial of the treatment in a case in which a temporary improvement had previously taken place after thyroid grafting.[73] The following month Gley made two injections in another case of myxœdema in a child nine years of age. All these four trials failed and in each case the treatment was abandoned. The want of success in these cases appears to have been due to the employment of too dilute a preparation of thyroid extract, rendered still less efficient by filtration through porcelain. The doses employed were too small and they were not repeated sufficiently often to give the treatment a fair trial. These results, however, were not published till later and the first suggestion of this treatment in France was published by Brown-Séquard and d'Arsonval in July, 1891,[74] at a time when the beneficial effects of the remedy had already been demonstrated in England.

Three months later Fenwick[75] published an observation on "The Diuretic Action of Fresh Thyroid Juice." He injected ten drops of freshly expressed thyroid juice mixed with an equal quantity of distilled water. In cases of myxœdema he observed an increase in the amount of urine for fourteen to twenty-one days. No other effect of the injections was mentioned, and the conclusion was drawn that myxœdema depended upon "a perverted renal function." During the next nine months several cases were published by W. Beatty,

E. Carter, and A. Davies, each of which illustrated the action of thyroid extract in removing the myxœdema. During 1892 several independent trials of the administration of thyroid extract by the mouth were made. The first trial of this simpler method of utilizing the remedy was made by Howitz,[13] of Copenhagen, who began to treat a case of myxœdema by feeding with the thyroid gland of the calf on March 22d, 1892. On July 6th, at the congress of Naturalists in Copenhagen, he stated that he had by this method obtained just as good a result as usually followed the injection of thyroid extract beneath the skin. In England E. L. Fox[14] began to treat a case of myxœdema by the internal administration of thyroid extract on June 2d, and on July 27th Hector Mackenzie[15] began to feed a case of myxœdema with fresh thyroid glands. The results obtained by these observers were published simultaneously on October 29th, and both clearly showed that the physiological action of the drug was obtained just as well when it was swallowed as when it was injected. In Scotland, Lundie[22] began to give thyroid extract by the mouth on July 16th with the same result. These observations have now been repeated and confirmed by many others. Thus an important advance was made in the manner of employing thyroid gland preparations which did away with many of the risks and inconveniences incurred by the employment of hypodermic injections. Thyroid extract should be given by the mouth and is now rarely injected. The details of the method of employing the extract will now be considered more fully.

Practical Application of the Thyroid Treatment.

The treatment of myxœdema in the great majority of cases is now carried out by the administration of the thyroid gland, or preferably of some preparation of the gland, by the mouth. The hypodermic method should only be used if it is desirable to produce a very rapid effect and the patient is unable to tolerate sufficiently large doses by the mouth. The remedy can be prescribed in various forms of which the following are the more important ones. The preparations referred to are made from the gland of the sheep.

1. The Thyroid Gland.—The thyroid gland itself may be given raw in doses varying from one-eighth of a lobe up to a whole lobe. The larger doses are apt to produce gastro-intestinal disturbance and other toxic symptoms. The portion of the gland should be minced and taken in glycerin or some other vehicle which will cover the somewhat nauseous taste of the raw tissue. The gland may be lightly cooked by frying or boiling before it is eaten. This process

does not entirely destroy the activity of the gland, though the large doses which have been taken with impunity after this mode of preparation appear to indicate that it diminishes it somewhat. It is much better, however, when possible, to employ one of the preparations which can now be so readily obtained.

2. Liquid Thyroid Gland Extract.—This preparation is a diluted glycerin extract of fresh thyroid glands prepared in the manner already described (*v.* p. 740). It consists of equal parts of glycerin, water, and liquid extracted from the glands. On the average the whole thyroid gland of one sheep yields six cubic centimetres or one and a half drachms of the extract. This is a most convenient preparation and fully active. It may be given in water in doses of three to twenty minims. It is best to prescribe the extract as it is prepared, and for the patient to measure out the dose which has been ordered, instead of prescribing it in a more dilute form in a mixture.

3. Thyroid Gland Powder.—There are several preparations of the thyroid gland in the form of powder. The gland itself may be dried at a low temperature and powdered or the juice of the fresh glands may be expressed, dried, and powdered. White[76] has prepared a powder consisting of the precipitate produced by the formation of calcium phosphate in the glycerin extract. Of this preparation about eighteen grains are obtained from each sheep's thyroid gland.

4. Thyroid Gland Tablets and Pills.—The preparations in the form of powder can be mixed with a suitable excipient and made into either tablets or pills. This is a convenient form for use when fresh supplies of the liquid extract are not easily obtained, as for example when the patient is travelling. Vermehren has employed the white precipitate, which is obtained by adding alcohol to thyroid extract, in the form of pills in doses from one and a half to four and a half grains. All these preparations have been proved to be active so that any one of them may be employed in the treatment of myxœdema. The choice of the form in which the remedy is to be given depends rather upon the circumstances and tastes of the patient than upon any special advantage that one form may have over another. I generally prescribe the glycerin extract, of which the patient obtains a fresh supply each fortnight. The doses mentioned in the rest of this section apply to the glycerin extract already described.

The treatment of a case of myxœdema may most conveniently be divided into two stages. This simplifies the description, for certain precautions which must be taken during the first stage are no longer necessary when the second is reached.

First Stage of Treatment.

During the first stage of the treatment our object is to remove the symptoms of the disease with as little risk and discomfort to the patient as possible. This stage is completed as soon as the patient is free from myxœdema. During the second stage we have, by preventing the return of the myxœdema, to keep the patient in the condition of health which has already been attained. This is easily managed by a modified form of the treatment employed during the first stage. In order to prevent disappointment it is important to explain the nature of the treatment to the patient at the commencement and to point out clearly that the improved condition of health can be maintained only by a continued use of the remedy for the rest of life, and that unless some thyroid preparation forms part of the patient's regular diet for the future the symptoms are certain sooner or later to return. This, however, is such a simple matter and involves so little trouble that it presents no real difficulty. The first stage of the treatment has to be carried out with great care in all cases in which the disease has lasted for some years, in the aged, and in those who show any indication of cardiac or arterial degeneration. During the last few years many cases of long duration have been successfully cured, but in the future such cases will be rarely if ever seen. In these cases it is always advisable to begin with small doses of the thyroid extract. If there are distinct signs of cardiac degeneration three minims of the extract or an equivalent dose of some other preparation once a day is sufficient. If this dose is well borne it may be increased to five and then to ten minims each day. This caution is necessary, as some cases are very susceptible to the action of the drug at first. Thus in one case three minims of the extract every other day was all that could be borne, and even this small dose when given more frequently soon accelerated the pulse to 120. After a time this patient was able to take five minims every other morning.

The patient should be kept at rest in bed for a time so that as little strain as possible may be thrown upon the heart and arteries. This is important, for patients who have suffered from myxœdema for a long time are quite unaccustomed to exercise, and as they improve under the treatment they are only too ready to avail themselves of their newly found strength and to exert themselves to a dangerous extent, unless they are confined to bed. The importance of this was very much impressed upon me by the death of two of my early cases from cardiac failure brought on by over-exertion. In one of these a lady of sixty-four, whom I saw in consultation

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with Dr. Walker, of Newcastle, had developed myxœdema five years previously. For about a year she had suffered from considerable dyspnœa on exertion and had had several attacks of syncope, one of which had nearly proved fatal, and probably would have done so had not medical aid been at once available. The heart sounds were weak and intermittent, but no signs of valvular disease could be detected. She passed safely through the first stage of the treatment and the myxœdema had practically disappeared, when one morning, while stooping to put on her shoes, she had another syncopal attack and died from cardiac failure before her medical attendant reached her. The other case was that of a woman, aged sixty-two, who had been myxœdematous for six or seven years and suffered from great cardiac dyspnœa on exertion. She also improved under treatment, but died suddenly one day from cardiac failure while trying to ascend a hill more rapidly than usual. Unfortunately in neither of these cases was a post-mortem examination made, but there can be no doubt that death was due to the degenerated condition of the heart. J. Thomson [22] has published a similar case in which, although the patient had been kept in bed, death took place when she sat up for a short time while the bed was being made. At the post-mortem examination the muscular substance of the heart was found to be in a state of extreme fatty and fibrous degeneration. In other similar cases this danger has been avoided only by keeping the patient in the horizontal position until recovery has taken place. Any signs of cardiac failure which may arise during this stage of the treatment must be promptly met by cardiac tonics, diffusible stimulants, and alcohol.

The change which is brought about by the treatment in the condition of a patient who is suffering from well-marked myxœdema is so great that it is not advisable to attempt to bring it about too quickly or the patient may suffer in consequence. Thus in one case of which I have heard, the extract was given in such doses that the patient's weight was reduced by two stones (twenty-eight pounds) in a fortnight, owing to the rapid diminution of the myxœdematous swelling, and an alarming state of prostration was produced in consequence. The time which is usually occupied in carrying out the first stage of the treatment varies from six weeks to three or four months, according to the condition and circumstances of the patient. In less advanced cases in which there is no cardiac or vascular degeneration it is not necessary to enjoin rest in bed, but no unusual exercise or excessive strain should be permitted until a cure has been effected.

In all cases the condition of the pulse is an important guide to the regulation of the doses. Any undue acceleration of the pulse amounting to ten or twenty additional beats in the minute indicates that the

dose is quite large enough. If any increase in the pulse-rate beyond this takes place the dose should be diminished either in quantity or in frequency. Thus if a dose of five minims of the extract is given each morning two or three hours after breakfast, and no distinct improvement has taken place by the end of a week or ten days, and the pulse has not been accelerated, the dose should be increased to ten minims, and later to fifteen if ten is not found to be sufficient. In early cases in which the heart and vessels are healthy ten minims may be given each day from the beginning. In some cases it is necessary to give as much as fifteen minims twice a day in order to rid the patient of her symptoms.

Other indications are the effect of the treatment upon the temperature and upon the alimentary canal. A rise of temperature amounting to a degree above the normal level indicates that the dose is too large. Any gastro-intestinal disturbance must be promptly checked by appropriate remedies and by lowering the dose of the thyroid extract. Foulis[22] has reported a case in which the patient died within twenty-four hours after taking a quarter of one lobe of a sheep's thyroid gland. Profuse diarrhœa set in which was rapidly followed by coma and death. Gastro-intestinal disturbance as indicated by vomiting and purging has not infrequently been produced by large doses of the raw gland. This, however, rarely occurs when preparations of the gland are used in moderate doses. The symptoms which may appear as a result of overdosing with thyroid gland, or with preparations made from it, are increased frequency of the pulse, elevation of the temperature above normal, prostration, vomiting, diarrhœa, headache, and sweating. Swelling of the parotid and submaxillary glands was observed in one case by Church[22] after an overdose of the gland itself. Such symptoms may be produced by a few unusually large doses or by taking a dose which is only slightly larger than necessary for a long period. As an example of this latter mode of overdosing I may cite a case in which during the summer, and long after all traces of myxœdema had disappeared, the frequency of the pulse was raised to 112, the skin was flushed and moist, and there was a fine tremor of the hands just like that which is often present in exophthalmic goitre. The symptoms rapidly disappeared when the dose was diminished. One of the most remarkable cases of poisoning by an overdose of thyroid gland has been recorded by Béclerc.[78] This patient took by mistake ninety-two grammes of thyroid gland in eleven days. This was followed by increased frequency of the pulse and respiration, elevation of temperature, insomnia, restlessness, polyuria, glycosuria, albuminuria, exophthalmos, temporary tremor of the arms, partial paraple-

gia, with a sensation of heat and sweating. The identity of many of these symptoms with those which occur in exophthalmic goitre is remarkable. This relationship will be considered in connection with the pathology of exophthalmic goitre.

Treatment by Hypodermic Injections.—If the hypodermic method is employed all the precautions which are necessary when the patient takes the extract by mouth are equally important. In addition to these certain other precautions must also be taken. The syringe must be one which can be easily cleaned and rendered aseptic. A syringe made with an asbestos plunger is suitable for the purpose. The syringe must be washed out several times with a five-per-cent. solution of carbolic acid before it is used. After use it must first be washed out with water and then with the carbolic acid solution again before it is put away. The injection should be made at some point where the skin is loose as in the interscapular region of the back. The skin must be carefully cleansed with the carbolic acid solution before the puncture is made. From five to fifteen minims may be injected at a time. The extract should be injected slowly at the rate of five minims a minute. Any sudden flushing of the face, pain in the back, or feeling of faintness indicates that the extract is being absorbed too quickly, probably owing to the point of the needle having entered a small vein. If this should occur, the injection must at once be stopped and the patient be made to lie down. A localized cellulitis and in some cases abscesses have been produced by the injections. During the first stage of the treatment the injections should be repeated twice or three times a week. When the second stage is reached an injection once a week or in some cases once a fortnight is all that is required. Some of my cases have been treated both by injection and by feeding, and the results show that taking one week with another about three or four times as much of the extract is required each week to produce the same effect by the mouth as by injection. Thus in one case a drachm of the extract taken in the course of a week in six daily doses of ten minims each appeared to be equivalent to a single weekly injection of fifteen minims. In another case a drachm taken by the mouth in a fortnight, in twelve daily doses of five minims each, was found to have almost the same effect as a fortnightly injection of fifteen minims. I never employ the subcutaneous method of administration now, as it is much better to give the extract by the mouth.

Second Stage of the Treatment.

During the second stage of the treatment our object is to prevent any return of the myxœdema by maintaining a sufficient supply of

thyroid secretion. As has already been pointed out, the thyroid gland still remains functionless though the myxœdema has been removed, consequently if the administration of thyroid extract is discontinued, the patient will sooner or later become myxœdematous again. The relapse under these circumstances takes place slowly but none the less surely. One of my patients at one time had no treatment for thirty-one days, during which interval the temperature fell to two degrees below normal and the swelling partly returned. At another time during a period of one hundred and two days she had only one hypodermic injection of twenty-five minims of thyroid extract on the eighteenth day; at the end of this time all the original symptoms had returned except that the newly grown hair had not been lost. On each occasion the symptoms rapidly disappeared when the treatment was renewed. By the regular administration of a sufficient dose of thyroid extract the patient can be kept entirely free from all symptoms of myxœdema as long as she lives. This I think is definitely proved by the result of the treatment in the first case in which it was tried. It is now more than four years since this patient was first treated with thyroid extract. For long she has had no symptom of myxœdema and is as well as she could be, both in mind and body, leading an active life as a working-man's wife. She takes one drachm of thyroid extract each week in daily doses of ten minims each.

At the commencement of the second stage of the treatment it is important to ascertain the most suitable constant daily dose for the patient. This varies in different cases, the difference depending partly on the patient and partly on the condition of the thyroid gland. The appropriate dose in any given case can be ascertained only by trial. It may vary from five to fifteen minims of the extract per diem, but as a rule lies between these limits. A single daily dose is preferable to larger doses at longer intervals, but this can be arranged according to circumstances to suit the convenience of the patient, who will readily appreciate the importance of the permanent continuance of the remedy. A fall of the temperature below normal, a slight return of the swelling or of any other symptom of the disease indicates that too small a dose is being given, just as acceleration of the pulse or any other sign of over-dosing shows that a smaller dose should be ordered. In a climate which does not undergo any great variations the same dose can be continued all the year round. In very hot weather a smaller dose suffices than in cold weather, and a dose which has been sufficient during the summer may be found to be too small to maintain the health during very cold weather. Otherwise when once the permanent dose has been settled the patient does not require to be seen

more than three or four times in the year. The second stage of the treatment is thus carried out with great ease, and the continuation of it gives but little trouble. In no case is it advisable to employ the hypodermic method during this stage.

The Results of Thyroid Treatment.

The results of the treatment of myxœdema carried out in the manner indicated are very striking, especially in well-marked cases. One of the earliest signs of improvement is a rise in the temperature. This is a sure indication that a sufficient dose is being taken to produce the physiological action of the drug and that improvement in other directions will soon follow. When the temperature has previously been two or three degrees below normal it usually reaches the normal level after a few days' to a week's treatment. A slight corresponding increase in the frequency of the pulse takes place at the same time, but, as already mentioned, any increase in the frequency out of proportion to the rise of temperature is to be avoided. The myxœdematous swelling diminishes and finally disappears. As a consequence of this there is a great improvement in the appearance of the patient as the face once more regains its natural expression. So great is this change that in some cases the patient has scarcely been recognized by her friends after the treatment.

If the reduction of the swelling is brought about rapidly, the skin for a time remains loose as a result of the previous stretching to which it has been subjected by the subcutaneous swelling. After a time it contracts and regains its normal elasticity. The movement of the limbs being no longer limited by the swelling becomes more free and active. Thus one of my patients could neither button his collar at the back of his shirt nor stoop to lace his boots, both of which he was able to do with ease after six weeks' treatment. The reduction of the swelling is accompanied by loss of weight which may amount to two or even four stones (28 to 56 pounds). Too rapid reduction of weight is to be avoided, as it is apt to produce prostration.

The skin becomes soft, smooth, and moist. During the earlier part of the treatment a certain amount of desquamation generally takes place. The skin as a rule comes off in small bran-like scales; in some cases, as for example in one recorded by Bramwell,[79] it peels off from the palms of the hands and the soles of the feet in large flakes just as it does after an attack of scarlet fever. Perspiration returns even when it has been absent for several years. The hair grows again after the treatment has been carried on for some months. In one of my cases (*v.* Figs. 46 and 47) in which the head was entirely bald,

except at one or two points where a few stray hairs still remained, the hair has grown well. This patient, who was placed under my care by Dr. James Angus, of Newcastle, had suffered from myxœdema for twelve years and before she lost her hair it had turned quite gray. The new hair is like that of a young girl. She is now fifty-six, but there are only a few gray hairs to be seen and some are now more than a foot in length. The hair in other parts of the body also grows. The nutrition of the nails improves also when the remedy has been taken continuously for several months.

The improvement in the general appearance of the patient is well shown in Figs. 44, 45, 46, and 47. The external signs are naturally the most striking, but the improvement in other respects is none the less important. When the temperature becomes normal the distressing sensation of chilliness which is so much complained of in cold weather also disappears.

The mental condition improves *pari passu* with the physical. The mental processes are once more performed with their normal rapidity, the memory improves, the hallucinations no longer trouble the patient, and the speech becomes distinct and fluent.

As an example of how complete the restoration to health may be, I may mention here the case of a gentleman (*v.* Figs. 73 and 74) who was sent to me by Dr. Fort, of Oldham. When I first saw him he was suffering from well-marked myxœdema and could hardly walk half a mile. After treatment by thyroid extract he recovered completely. He also required a daily dose of ten minims of the extract during the second stage of the treatment. He subsequently was able to undertake the duties of secretary to a large manufacturing company, and two years after the treatment was first commenced he was able to ascend a mountain over three thousand feet high, including a walk of ten miles without feeling undue fatigue.

In some cases in which actual insanity has occurred as a result of the myxœdema the insanity has been cured. Striking examples of the power of thyroid extract to cure really bad cases of the insanity of myxœdema have been published by Claye Shaw, Beadles,[80,84] and Dunlop.[22] These cases have been cured rapidly by treatment lasting from two to seven months. In other instances great improvement in the mental condition has taken place as a result of the treatment. In the future, if all cases of myxœdema are treated as soon as their true nature is recognized, we may reasonably expect that the insanity of myxœdema will cease to exist. The bodily activity and muscular strength return, and as a result of this, combined with the improvement in the personal appearance, the patient is no longer content to sit quietly at home as before, but soon begins to go about again as

usual. The number of red corpuscles in the blood increases as a rule, as in a case recorded by Sir T. Grainger Stewart. In patients who have not passed the climacteric, menstruation returns and takes place regularly even when there has been amenorrhœa for several years. Many cases of myxœdema have, however, already passed the menopause at the time the treatment has been applied, and in such no return of menstruation must be expected.

The quantity of urine passed during the earlier stages of the treatment is distinctly increased in some cases. In many cases the treatment has been carried out under circumstances which did not permit of an accurate measurement of the urine being made, so that no reliable data are available as to the actual frequency of diuresis as a result of taking thyroid extract. In my own cases I have not been able to trace any definite diuretic action to thyroid extract. The amount of urea in the urine is increased while the patient is taking thyroid extract, as has been shown by T. Oliver.[81] A careful determination of the amount of nitrogen taken in the food and of the total nitrogenous excretion both before and during the administration of thyroid extract has been made in one case by Ord and White.[82] They found in this case that the urine was increased in volume and that the nitrogen excreted in it exceeded the total quantity of nitrogen in the food. The increased nitrogenous excretion was found to be chiefly in the form of urea; the phosphoric acid and chlorine elimination were, however, practically unaffected. In one of my cases[30] albuminuria which had been present for some time disappeared when the myxœdema was cured. In short we may say, provided no incurable degeneration of an important organ has taken place, that myxœdema can be entirely cured by thyroid extract even when the disease has already lasted several years. I say cured advisedly, for though it is true that the chronic interstitial thyroiditis still remains, the myxœdema is certainly cured.

CRETINISM.

(Crétin, Swiss patois for Chrétien, a Christian; or Latin creta, chalk.)

Synonyms.—Cretinoid Idiocy. Latin: *Cretinismus;* German: *Cretinismus;* French: *Crétinisme, Idiotie avec Cachexie Pachydermique, Pachydermie Crétinoïde.*

Definition.

The term cretinism has not infrequently been used somewhat vaguely so as to include several conditions which are pathologically

quite distinct. Here, however, we shall use it only to denote a condition which is due to diminished functional activity of the thyroid gland commencing before birth or in early life before the age of fifteen. True cretinism is therefore a congenital or infantile form of myxœdema. The disease may, however, commence later than infancy as at eight or twelve years of age, so that we have a series of rare intermediate types between congenital cretinism and adult myxœdema. When the symptoms of myxœdema develop early we always find, in addition to the symptoms observed in the adult, an arrest of mental and bodily development at the age at which the disease commenced. Thus two cretins of the same age may differ considerably in development if the disease began during the first year of life in the one and at eight years of age in the other. Cretinism may be endemic, as it is in certain parts of the Continent, or sporadic, as it is in England. Some cretins are goitrous and others have no goitre. The sporadic cases as a rule have no goitre, the thyroid gland being either undeveloped or atrophied, while one-third of the endemic cretins also have no goitre. In both cases the functional activity of the thyroid gland is much diminished by disease, which in the one class has led to atrophy and in the other to the formation of new tissue in the gland, so that with the exception of the size of the gland the two conditions are identical.

Congenital Cretinism.

It is rare to find the characteristic features of cretinism developed before birth. Some such cases have been recorded as examples of intra-uterine rickets. The child seldom lives, and in all the cases which have been examined the thyroid gland has been found to be either undeveloped or to have undergone complete atrophy. The body appears short and broad. The peculiar elastic swellings or tumors which occur so frequently in other forms of cretinism are present in each supraclavicular fossa. The skin is thick and often lies in folds. The subcutaneous adipose tissue is well developed. The head is short and broad, the cranial bones being thick and the facial bones ill developed. There may be ossification of the basi-sphenoid junction while the other sutures remain open. All the bones of the skeleton are short and broad, while the epiphyses are swollen but not ossified. Intermediate between this form of cretinism and the more usual form which develops during infancy, Horsley [85] describes a type in which the disease commences shortly before birth but develops slowly, so that at the time of birth the disease is not nearly so much advanced as in the form we have just considered,

and the child is able to live. In this type there is usually a goitre at birth. The body appears to be large and there may be a myxœdematous swelling of the subcutaneous tissues. The nose is flat, the tongue large, and the neck short and thick. Mental development is slow. Later in life the symptoms become more developed and a severe form of cretinism results from the progressive degeneration of the thyroid gland.

Sporadic and Endemic Cretinism.

In this form of cretinism the child is perfectly healthy at birth and may continue to develop in a normal manner for several months or for two, three, or four years before any sign of the disease is noticed. The disease may commence still later, and we shall consider all cases in which the symptoms appear before the age of fifteen as examples of cretinism.

Etiology.

We know that sporadic cretinism is the result of disease of the thyroid gland just as much as adult myxœdema is, but we do not know what the exciting cause of the change in the thyroid gland may be. Certain circumstances appear, however, to act as predisposing causes.

Age.—The disease most commonly develops during the first three or four years of life, though in exceptional instances it may not commence until the eighth or even the twelfth year.

Sex.—The female sex is more liable to cretinism than the male. Bramwell[8] has tabulated 44 cases, of these 30 were females and 14 males. Langdon Down referred in his Lettsomian lectures to 11 cases, of which 3 were males and 8 were females. I have collected from recent literature and from information kindly given me by my friends, the records of 52 additional cases, some of which I have had the opportunity of observing. Of these 34 are females and 19 males. If we add all these together we obtain a total of 108 cases, of which 72 are females and 36 males, a proportion of two to one. Fletcher Beach[86] has collected 100 cases in addition to 16 which have been under his own care and finds that there were not quite twice as many females as males among the 116 cases. Thus we see that the disease is twice as frequent in the female as in the male, a much nearer proportion than in the adult, in whom we have seen that the disease is nearly seven times as frequent in the female as in the male.

Heredity.—Direct heredity does not appear to take part in the production of sporadic cretinism. Indeed, it could scarcely do so, seeing that in cretins the sexual functions are practically in abeyance. Bramwell has found that in a considerable number of cases

the parents have either been neurotic or have suffered from some form of tuberculosis. The parents of sporadic cretins are often living amid unwholesome surroundings and upon inferior food. Langdon Down [87] considers that alcoholism in the parents, especially if it exists at the time of procreation, is an important factor. The disease may occur in several members of the same family and Bramwell [8] quotes a remarkable instance recorded by Stirling in which three girls and one boy, in a family of eleven, were cretins. Heredity plays an important part in the production of endemic cretinism, for cretinism often develops in the children or grandchildren of those who have suffered from endemic goitre. Fagge [88] mentions the interesting fact that it has been noticed that if a family migrates into a district where goitre and cretinism are both endemic, goitre develops first, and cretinism only in the second or third generation.

Locality.—Unlike endemic cretinism, the sporadic form is not confined to special localities. Cases have been recorded in England, Scotland, Ireland, France, South Australia, and America. Bramwell and J. Thomson [22] have both noticed that sporadic cretinism and myxœdema are especially prevalent in one part of Edinburgh. Endemic cretinism occurs in certain localities in which goitre is also endemic, and it would appear that both are due to the same local condition. Endemic cretinism occurs most frequently in the shut-up valleys of mountainous districts, in various parts of Europe, Asia, and America. What the actual local condition which produces the disease may be has not yet been ascertained. Endemic goitre is commonly attributed to some constituent of the drinking-water which is rendered harmless by boiling. It is probable that this also may be the direct or indirect cause of endemic cretinism. In England the disease was prevalent in Chiselborough in Somerset, in 1847, as 24 out of a population of 350 were cretins, but it has since died out. More recently Robinson [89] has pointed out that a certain number of cretins are to be found in the north of England in the upper valleys of the Pennine Range, as at Alston, Blanchland, and Stanhope where goitre is endemic, so that in this district cretinism may still be said to be endemic, otherwise only the sporadic form is now to be found in England. Sporadic cretinism occurs in America as it does in other parts of the world, but Osler [93] shows that there is little evidence that it is endemic in any part of America.

Symptoms.

In considering the symptoms of sporadic cretinism we shall describe them as they are usually seen in cases in which the disease has commenced in infancy or before the fifth year of life and in which

they become well marked by the time the child has reached the age of twelve or fifteen years, if not earlier. The child is quite normal at birth and remains healthy until the thyroid gland begins to be affected. It is then noticed to be less lively than before, and the growth and general development gradually become arrested. The symptoms then slowly develop until the child reaches the condition which we are about to describe. The whole body appears to be short and stunted, as but little further growth takes place after the onset of the disease. The subcutaneous tissues are somewhat swollen by a solid œdema, which is most noticeable on the eyelids, hands, and feet. The complexion is of a dull, chalky color. The eyes appear to be rather widely separated, while the nose is broad, flat, and somewhat turned up at the end. The cheeks are baggy and the lips thick, the lower one being often everted. The mouth is large, the tongue is thick and big and is frequently protruded from the mouth. The head is narrow in front but broad behind, the anterior fontanelle remains unclosed, and the ears are large. The whole aspect is ugly and in some cases extremely repulsive. The skin is dry and rough and the hair coarse though it may be fairly plentiful. The scalp is often eczematous. The teeth appear late and are often carious. The neck is short and thick, while the supraclavicular fossæ are occupied by the elastic swellings which are commonly called "fatty tumors." The abdomen is large and prominent, and either an umbilical or an inguinal hernia is generally present. The lower dorsal and lumbar spine is often considerably arched forward. The limbs look short and broad in consequence of the arrest of their natural growth. The hands and feet are broad and thick. The temperature is generally rather below the normal. There is marked anæmia. Lebreton and Vaquez[94] found in one case that before treatment the red corpuscles were larger than usual, but that after treatment by thyroid glands the corpuscles were normal in size. They also found some nucleated red corpuscles in the blood. They conclude that this resemblance of the red corpuscles to those of the fœtus indicates that the blood, like other tissues, is arrested in development. The voice is harsh. The gait is slow and awkward. The expression is dull and apathetic, being entirely wanting in the bright interestedness of the healthy child. The mental condition is one of dull placidity with contentment, while there is a great dislike to bodily exertion of any kind. Cretins sleep well and often for much longer periods than healthy children of their own age. The mental capacity varies considerably in different cases, as some are quite helpless idiots, while others can feed themselves and appear to be as intelligent as most children of three or four years.

Cretinism in the Adult.

A certain number of both sporadic and endemic cretins live to become adults and may survive until they are thirty or forty years old. In these cases a small amount of slow development may continue after the onset of the disease up to the twentieth year, after which there is but little further change during the rest of life. Cretinism in the adult presents the same features, whether it be sporadic or endemic in origin, so that the following description applies to either form. It must, however, be remembered that about two-thirds of the endemic cretins are goitrous, while the rest, like sporadic cretins, have no goitre.

Symptoms.

The skeleton is short and growth may be so much arrested that adult cretins not infrequently measure only three feet or even less in height. The following table of measurements of a male cretin, thirty-two years old, published by Lloyd,[95] of Philadelphia, gives a good idea of how small the skeleton may remain.

Height		35.5	inches.
Head:	Fronto-occipital diameter	7.5	"
	Bitemporal "	5.5	"
	Biparietal "	5.5	"
	Circumference	22.25	"
	From ear to ear	12.25	"
	From root of nose to occipital protuberance	13.5	"
Limbs:	From ant. sup. spine of ilium to int. malleolus	14.5	"
	Length of femur	7.	"
	" humerus	5.5	"
	" radius	5.5	"

The following table gives the measurements of a female cretin, 28 years of age, whom I saw in consultation with Dr. Bunting, of Scotswood. She is pictured in Fig. 48:

Height		34.5	inches.
Head:	Circumference	21.5	"
	From ear to ear	13.5	"
	From root of nose to occipital protuberance	13.5	"
Limbs:	From ant. sup. sqine of ilium to int. malleolus	15.	"
	Length of femur	9.	"
	" humerus	5.5	"
	" radius	4.5	"

The head is broad and short, and Horsley [85] mentions that wormian bones are often found in the sutures of the skull. The anterior fontanelle may still remain widely open. The forehead is low and

receding, the eyes are widely separated, and the nose is broad. The amount of myxœdematous swelling varies; in some it is very slight, in others it is well marked, as in the ordinary adult form of myxœdema. The spine may be deformed by angular or lateral curvature and there is usually more or less lordosis. The skin is coarse and rough and brown in color. The hair which may be fairly plentiful on the head is thick and coarse. The nails are rough and thickened. The teeth are defective. The mouth is large, the lips are swollen, and the tongue is large and often protruded. The abdomen is large and prominent. The lower limbs are often wasted but the hands and feet are broad and thick. The functions of the body generally are performed sluggishly. Voluntary movements are executed slowly and deliberately. Respiration and pulse are slow, and the temperature is subnormal. The generative organs are generally like those of a child and the sexual functions are in abeyance. The mental condition depends chiefly upon the severity of the general symptoms and varies between complete idiocy and a moderate amount of intelligence with capability of undergoing improvement by suitable education. The temperament is placid and indolent. Speech is usually defective. Of the special senses hearing is most frequently imperfect, and it was stated by the Sardinian commission to be affected in two-thirds of the cases examined. The sense of smell is also deficient, but sight as a rule is unaffected.

FIG. 48.—Sporadic Cretinism in a Patient, aged 28 years; height, 34½ inches.

Pathological Anatomy.

The observations upon the morbid anatomy of cretinism are few in number. Of the 44 sporadic cases tabulated by Bramwell,[8] 10 were examined post mortem. In the 9 cases in which the condition was noted, the thyroid gland was found to be absent. Enlargement of the hypophysis cerebri was found in 5 cases by Nièpce[41] and by Boyce and Beadles[36] in 1. Horsley[85] mentions that in addition to these changes the convolutions of the brain are ill-defined and the blood-vessels are small in proportion to the rudimentary condition of the nervous system.

Pathology.

We have already considered the pathology of myxœdema in the adult at some length, and to avoid repetition we would refer the reader to that part of the article, in which will be found the reasons for concluding that all forms of myxœdema are due to loss of function of the thyroid gland. When thyroidectomy has been performed in young animals it has been found to lead to arrest of development as well as to the production of myxœdema, in fact to a condition of cretinism. Hofmeister found that removal of the thyroid gland in young rabbits is followed by a remarkable arrest of development, the growth of the long bones especially being affected. He has also observed roughness of the skin, loss of hair, and swelling of the abdomen. Horsley[6] has shown that by placing monkeys after thyroidectomy in a warm room with a constant temperature of 90° F., life is prolonged considerably; the average duration of life after the operation being one hundred and twenty-five days instead of only twenty-four days, the usual duration when the animals are kept at ordinary temperatures. Animals thus treated acquired all the characters of a cretin. During the first few weeks slight attacks of tremor and malaise were observed. The animals became dull and inactive with occasional manifestations of idiotic activity. The posture usually assumed was that of a cretin. Rapid wasting took place at first, although the animals consumed large quantities of food. Many of the symptoms of acute myxœdema as already described were also observed. Much of the hair was lost and the voice gradually became very hoarse. The animals ultimately died comatose.

In man complete cretinism may occur as a result of thyroidectomy at an early age, just as the adult form of myxœdema may follow the same operation performed after full development has taken place. One case published by P. Bruns[96] and recorded in the report of the

Clinical Society is of special interest from the light it throws upon the pathology of cretinism. In 1866 when the patient was ten years of age Sick removed an entire goitre, with the result that when he was examined eighteen years later by Bruns he had become a dwarfed adult cretin. Bodily development had been arrested from the time of the operation. The whole face, and especially the lips and lower eyelids were swollen, and the expression was idiotic. The skin was dry and the hair scanty. The mental capacity was less than that of a boy ten years old. The speech was slow and he was incapable of doing any work. In this case no trace of thyroid tissue could be discovered either before or after death. At the post-mortem examination the border of the epiphysis was easily recognized on the humerus and also on the proximal end of the femur, while the epiphysis of the trochanter major was still cartilaginous. Further evidence that cretinism is due to loss of function of the thyroid gland and to the absence of thyroid secretion from the blood is afforded by the results of the treatment of cretinism by thyroid extract.

Treatment.

Cretinism like myxœdema was until quite recently considered to be a hopelessly incurable disease and only palliative measures were attempted, such as keeping the patient warmly clad and in a warm atmosphere. Jaborandi or pilocarpine was employed when the skin was very dry. In considering the treatment of myxœdema we have already dealt with the theory and development of the treatment by thyroid grafting or by the internal administration of thyroid extract, so that here we shall consider only the special application of the treatment to cretinism.

Thyroid Grafting.—The details of this operation are the same as in the case of the adult, and it should be preceded by a prolonged course of treatment with thyroid extract as has been suggested by Horsley.[70] In several cases this treatment has been followed by very considerable improvement in the condition of the patient for a time, but afterwards the symptoms tend to return owing to atrophy or absorption of the grafted gland. J. L. Gibson[97] grafted two lobes of a lamb's thyroid gland on two separate occasions into a cretin six years old. On each occasion improvement followed. After the second operation the patient became more intelligent and active; the swelling almost entirely disappeared, the skin became soft, and he grew two inches in twelve months. If it be proved that a graft can be made to take after a course of treatment by thyroid extract, this will no doubt be the best method of treating cretinism, as it will obviate the necessity of

the continuous taking of the extract for the whole of life. At present, however, the best results are obtained by the continuous administration of thyroid extract.

Thyroid Extract.—In the treatment of a cretin we have a somewhat prolonged and formidable task to perform. If the case comes under observation early in life soon after the disease has developed, we have in the first stage of the treatment to remove the actual symptoms of the disease. This may be accomplished in from three or four months to a year. In the second stage of the treatment we have not only to give enough thyroid extract to prevent the return of the symptoms, but a sufficient quantity to supply the growing needs of the child and to enable him to continue his normal growth and development. In cases in which the condition has already lasted many years we have to remove the actual symptoms of the disease and to help the patient to renew his development from the point at which it had become arrested. This is naturally a more difficult matter in adult cretins than in cases in which the treatment is commenced in childhood, yet we shall see that a most remarkable change can be brought about even in adult cretins in a comparatively short space of time.

As all cretins are small from arrest of development they must be treated as children, even though they may be adults in years. Relatively larger doses of thyroid extract can be given with impunity to cretins than to myxœdematous adults. Even cases of long duration can be treated without being confined to bed, as there seems to be little if any liability to syncope from exertion during the treatment. It is advisable, however, not to allow any unusual exercise to be taken during the first part of the treatment. It is a good plan to begin the first stage of the treatment with small daily doses which can be increased if they are well borne. In a small cretin, from one to three minims of thyroid extract each day is a sufficient initial dose. This can be increased to five, seven, or more minims each day or every other day if necessary. The extract is the most convenient preparation to use, as the dose can be exactly measured. Some cases have, however, been successfully treated by giving from an eighth to a quarter of the lobe of a raw sheep's thyroid gland twice a week. The raw gland is liable to set up toxic symptoms unless it is given in small doses.

As time goes on and the cretin grows, a tolerance of the drug appears to be established, and it is often necessary to increase the dose from time to time according to the progress of the case. If too large a dose is given, increased frequency of the pulse, pains, restlessness, headache, too rapid rise of temperature, or purging may follow.

The second stage of the treatment lasts for the whole of the patient's life. It is very easily carried out by the patient taking a suitable dose of thyroid extract each day or every other day, or twice a week if preferred. A regular daily dose is best, as it becomes a matter of routine and is less likely to be omitted.

Results of Treatment.

The results of the treatment of cretinism are remarkable; the most sanguine believer in the efficiency of medicinal treatment would, a few years ago, have been incredulous had he been informed that cretinism was curable by the simple administration of a drug, but curable it certainly is, as the results of treatment have already shown. The improvement in the physical condition is more rapid and appears earlier than the mental improvement. The swelling gradually diminishes in all parts of the body so that the face becomes thinner and more natural in appearance. The lips decrease in size and the tongue becomes smaller so that it is no longer constantly protruded, and the mouth can be closed. The blueness of the tongue and lips also diminishes. The alæ nasi become narrower. The supra-clavicular swellings diminish in size and finally disappear. The abdomen becomes less prominent and any herniæ which may be present diminish in size and may finally disappear altogether. The limbs appear to waste at first, but they grow stouter again afterwards. There is a decrease of weight while the swelling is diminishing, afterwards the weight increases owing to the deposit of normal adipose tissue and the renewed growth of the body generally. The skin becomes smooth and moist, the chalky tint of the complexion disappears, and the anæmia diminishes. The skin desquamates and may peel off the palms of the hands and the soles of the feet in large flakes. The hair grows and the anterior fontanelle diminishes in size, owing to the progressive ossification of the surrounding bones. The appetite improves, new teeth of the permanent set are cut, and the bowels act regularly. The temperature gradually regains the normal level, and further evidence of the general increase in metabolism is given by the increase in the quantity of urea which is passed in the urine, as has been shown by W. W. Ord[101] and by Railton.[61] In female cretins who are old enough, menstruation appears in due course. Muscular power increases so that more bodily activity becomes possible and soon shows itself by more active movements.

One of the most remarkable results of the treatment is the renewed growth in height which takes place. Thus G. E. Anson[98] observed in a child ten years of age and 40 inches in height a

growth of 4 inches in one year, when the height had increased only by 2 inches in the two years preceding the commencement of the treatment. In J. Thomson's [99] patient, aged eighteen years and eight months, 33½ inches in height, after growth had been arrested for fourteen years there was an increase of 4⅜ inches in the height in twelve months. In Bramwell's [100] patient, aged sixteen years and four months, 29½ inches in height, the growth amounted to as much as 6½ inches in six months.

The mental improvement is slow at first but is none the less remarkable. By degrees the usual shyness disappears, new words are learned, and speech becomes more ready. More and more intelligence is displayed and also a capability of enjoying life which forms a striking contrast to the previous dull and miserable hebetude. All these signs of improvement have been observed already in the short time during which the new treatment has been employed. From this remarkable progress we may fairly conclude that, if a cretin is put upon some form of regular and continuous thyroid treatment or diet as soon as the disease develops, he has a good prospect of growing up and of developing into a healthy adult in due time—provided always that the treatment is adequately carried out without intermission. If this is done in all cases we should not in the future see anything of that "pariah of nature," the adult cretin.

When a cretin has already passed the age of puberty without treatment, we can scarcely expect such good results as when the treatment is commenced in early childhood; nevertheless the results which have been obtained in cretins, sixteen and eighteen years of age, show that an adult cretin is capable of undergoing very great improvement and of renewing his development. In the case of a cretin, aged twenty-three, who was sent to me by Dr. Gibbon, of Tyne Dock, the disease first began to develop at the age of twelve years, and no further growth took place after fourteen. When I saw him first he measured 4 feet 7½ inches. Although growth had been arrested for nine years he began to increase in height again as soon as he commenced to take thyroid extract. Time only will show what amount of improvement an adult cretin may be capable of, and it would be rash to speculate until longer experience shows us whether the rapid rate of improvement and development taking place at the commencement of treatment will be continued. In those countries in which cretinism is endemic it is clearly the duty of the state to provide the treatment under competent supervision for the poor, as the cretins might be made useful members of the community instead of the useless and often helpless idiots they become if untreated.

EXOPHTHALMIC GOITRE.

(ἐξ, out; ὀφθαλμός, the eye.)

Synonyms.—Graves's Disease; Basedow's Disease; Cardio-thyroid exophthalmos (Walshe); *Struma exophthalmica; Tachycardia strumosa* (Lebert); German: *Basedow'sche Krankheit; Glotzaugenkropf; Glotzaugencachexia* (Basedow); French: *Maladie de Graves; Goître exophthalmique;* Italian: *Morbo di Flajani.*

Definition.

Exophthalmic goitre is characterized by five primary symptoms. These are enlargement of the thyroid gland, increased frequency of the pulse, protrusion of the eyeballs, fine tremor, and general nervousness. In addition to these there may be a considerable number of secondary symptoms, changes in the general condition of the skin, nervous system, alimentary canal, respiratory and genito-urinary organs. Of the primary symptoms the exophthalmos may be absent, and the enlargement of the thyroid gland may be so slight as to escape observation. Tachycardia and the nervous excitability appear to be present in all cases. The pathology of the condition is by no means clear. By many it is considered a disease of the nervous system. Be this as it may, much evidence has now been accumulated to show that many of the symptoms are referable to over-activity of the thyroid gland. Of the cause of the change in the thyroid gland we are as yet ignorant.

History.

Exophthalmic goitre has been recognized as a distinct disease for the last sixty years. During this period its peculiar nature has led to many and varied discussions, from which as a natural consequence has sprung a very copious literature. The various monographs and papers which have been written concerning this interesting condition now number several hundreds. In England the first description of the disease has been ascribed to Graves, and in Germany to Basedow. Thus in the former country and in France it has commonly been called Graves's disease, while in other parts of Europe and especially in Germany Basedow's disease is the designation in general use. Each observer described the disease independently, but the evidence that Graves published his description first is quite clear. The question has recently been dealt with at considerable length by P. Mannheim,[116] who clearly shows that Basedow's name should not be applied

to the disease at all, although Hirschberg [117] in another recent publication still maintains the priority of v. Basedow. It is, however, preferable to employ the name exophthalmic goitre which involves no question of priority of description. During the earlier part of the nineteenth century several cases were published by different authors in which exophthalmos was a prominent symptom. Some of these probably were cases of exophthalmic goitre; if so it is evident that the relationship of the primary symptoms to each other was not recognized by the writers who recorded them. The earliest record of a case of undoubted exophthalmic goitre is unfortunately by an anonymous writer.[102] In this case, which is referred to by W. Begbie,[103] a young lady, twenty-two years of age, suffered from palpitations. She was nervous and plethoric. There was a swelling the size of a goose's egg on each side of the neck and the eyes were very prominent. This was evidently a case of exophthalmic goitre, though we do not know whether the writer recognized the combination of symptoms as evidence of a distinct but undescribed disease or not.

In 1825 C. Parry published several examples of a disease which had not previously been described in medical writings. These were evidently cases of exophthalmic goitre. One was the case of a young woman, twenty-one years of age, who after being much frightened by a fall, was seized with palpitations and became very nervous. A fortnight later a goitre developed. The pulse ranged from 96 to 108 and there was strong pulsation in the carotid arteries. In 1828 Adelmann [104] published a case which has been considered by Virchow to be an example of exophthalmic goitre and consequently the first described in Germany. In this case there was strong cardiac and epigastric pulsation associated with goitre, and a rapid pulse varying from 106 to 160, over which digitalis exercised no influence. Adelmann called attention to the interesting nature of the case, but appeared to draw no conclusions from it. In 1833 Trousseau [105] gave an account of a woman, "affectée de goître et d'exophthalmie en même temps que de palpitations cardiaques," but as he says in a later work: "A cette époque j'étais loin de supposer que la triade symptomatique dût constituer une espèce pathologique, une entité morbide."

In 1835 Graves [106] published his well-known clinical lecture on account of which his name has usually been associated with the disease in England. His description of the disease was repeated in nearly the same language in his "System of Clinical Medicine" in 1843. In the lecture he described three cases in which there was palpitation of long duration without organic heart disease. There was also enlargement of the thyroid gland which was increased dur-

ing the attacks of palpitation. In one case, that of a woman aged twenty, the pulse was 100, and later both exophthalmos and goitre developed. Graves was the first to draw attention to the essential relationship between the enlargement of the thyroid gland and the palpitation without cardiac disease. In 1840 von Basedow [107] published the results of his observations on four cases of exophthalmic goitre. In this publication a more exact account of the disease was given than in any previous paper, but though the description was the result of von Basedow's own observations, his name should not be attached to the disease which had already been described by Graves in 1835. Three of von Basedow's cases were women and the fourth a man. In each of them were present the three cardinal symptoms to the combination of which von Basedow drew attention more emphatically than others had done previously. In these cases he also described perspiration, sensations of heat, diarrhœa, dyspnœa, and in three of them restlessness and hurried speech.

Von Basedow gave the name exophthalmic cachexia (*Glotzaugencachexia*) to the disease. After von Basedow's publication the number of cases reported steadily increased. Among those who recorded cases may be mentioned Romberg,[108] who carefully described six cases in 1851; Schoch, who collected thirty-four cases in 1854; and Koeben,[109] who first referred the disease to changes in the sympathetic nervous system in 1855. Stokes, in a book on diseases of the heart published in 1855, devoted a chapter to exophthalmic goitre, in which he described the "increased action of the heart and of the arteries in the neck, followed by enlargement of the thyroid gland and eyeballs." He drew special attention to the nervous phenomena of the disease.

Charcot [110] gave a very thorough description of the disease in 1856 and 1857, and again the following year. He laid great stress on the presence of palpitations which always developed before the exophthalmos and the goitre. In 1857 von Graefe [111] described the affections of the cornea and referred to the insufficient covering of the eyeball and the consequent lack of moisture. He considered that the prognosis was more favorable in cases in which the pulse-rate did not exceed 120 than in those in which there was a greater frequency. Fischer,[112] in a comprehensive description of the disease published in 1859, came to the conclusion that the symptoms were remote effects of anæmia. In a paper on exophthalmic goitre, published in 1860, Trousseau [113] contended that the cause of the symptoms was to be sought for in a diseased condition of the nervous system and that the disease should be classed as a neurosis. In the same year Aran [114] asserted that the sympathetic was affected and that the exophthalmos

was due to a contraction of the muscular fibres of Müller which were supplied by the sympathetic. Two years later a most important discussion upon the disease took place in the Academy of Medicine in Paris.[115] Both Trousseau and Piorry took a prominent part in this discussion. Trousseau then maintained that in all cases of exophthalmic goitre there was either an antecedent or a coincident special condition of the heart. He agreed with Aran in his classification of the disease as a neurosis. Piorry denied the existence of any special unity of the disease. He considered that the prominence of the eyeballs was a result of retarded intracranial circulation caused by the pressure of the enlarged thyroid gland upon the external and internal jugular veins. He also held that the goitre was the essential feature of the condition and that it led to a change in the blood and to respiratory troubles by which pathological changes in the digestive and generative organs of the female were brought about.

During the last thirty-three years a great number of publications dealing with exophthalmic goitre have appeared. Space will not permit of their separate consideration, but the more important papers will be referred to in the following pages.

Etiology.

In considering the etiology of exophthalmic goitre we have to consider first the predisposing causes and the circumstances under which the disease most frequently arises, and then the exciting causes to which the actual commencement of the illness can be traced in some cases.

Age.—The earliest age at which exophthalmic goitre has been observed is two and a half years. In the case of this girl which was recorded by Deval, the disease came on after scarlet fever. Charcot has alluded to a case in a man sixty-eight years old. Thus the disease may occur at any age between these two extremes; but the most usual period is between the ages of fifteen and fifty, and within this limit a greater number of cases develop between the ages of twenty and thirty than in any other corresponding period. Bramwell[118] considers that the most common period for the commencement of the disease varies in the two sexes, extending from fifteen to thirty years of age for women, and from thirty to forty-five years of age for men. In Eshner's table of 227 cases quoted by Pepper[119] the average age was between thirty and thirty-one years.

Sex.—There is a marked difference in the frequency of exophthalmic goitre in the two sexes, as women are much more liable to suffer from it than men. In men the disease is rare, but von Graefe

states that in them the disease tends to be more severe and acute than in women. It is variously estimated by different authors as being from two to ten times as frequent in women as in men. The following table of 400 cases which have been collected by several writers whose names are given, includes 22 cases which have been under the writer's own observation.

	Males.	Females.	Total.
Bryson	8	22	30
Caracoussi	0	6	6
Cheadle	1	30	31
De Ranse	3	9	12
Lendon	1	6	7
Lewin	3	24	27
Mackenzie	1	30	31
Mannheim	3	44	47
Maude	0	15	15
Murray	1	21	22
Praël	1	28	29
Putnam	9	25	34
Rosenberg and Henoch	4	23	27
Russell Reynolds	1	48	49
Schnitzler	2	6	8
Taylor	5	20	25
Total	43	357	400

Of these 400 cases 43 are males and 357 females, so that the disease occurs 8.3 times as frequently in the female as in the male sex.

Heredity.—Exophthalmic goitre is not often directly inherited, but there is a sufficiently large number of cases on record which have occurred in one or more of the near relations of the patient to show that in some families there is a distinct tendency to suffer from the disease. Mackenzie [120] mentions a most remarkable example of this which has been recorded by Oestereicher in a family in which the mother was hysterical, and eight out of her ten children suffered from varying degrees of exophthalmic goitre. One of the children had four granddaughters also affected with the disease. Mackenzie also mentions six pairs of sisters who have been affected. J. Rosenberg [121] records a case in which one sister, the father, two paternal aunts, and the paternal grandmother of the patient all suffered from the disease. Eulenburg and Thyssen have each seen mother and daughter, and Edwards father, mother, and daughter suffering from exophthalmic goitre. Three sisters; two sisters, a cousin, and an aunt; and a mother and three sisters have been recorded as suffering from the disease by Hale White, Cheadle, and Wild respectively. Corning reports an instance in which the malady was

observed in three successive generations. In some families there is a distinct tendency to suffer from diseases of the thyroid gland. Thus one member of the family may suffer from exophthalmic goitre while another is myxœdematous. In one family which has been observed by Maude [122] the mother suffered from myxœdema, one son had a slightly enlarged thyroid gland, and one daughter had a large goitre for some years and then developed exophthalmic goitre. I have seen one family in which one sister, aged twenty-six, had well-marked exophthalmic goitre. The thyroid gland had been enlarged as long as she could remember and the right lobe was rather larger than the left. There were a slight exophthalmos, general nervousness, dark-brown pigmentation of the skin, and a pulse of 160. Another sister, aged thirty-six, had an enlarged thyroid gland which had been in that condition as long as she could remember; the skin was dark, and the pulse 108. A third sister, aged eighteen, also had an enlargement of the thyroid gland which had been present as long as she could remember; the skin was dark in color and the pulse 100, but she was pregnant at the time. These three sisters came to see me together, and they informed me that a fourth sister also had a goitre and that the mother had had a swelling in front of her neck which was probably also an enlarged thyroid gland.

Exophthalmic goitre often appears in families which inherit a tendency to suffer from various diseases of the nervous system, such as epilepsy, hysteria, chorea, and insanity. Different forms of nervous diseases may occur in several successive generations of the family or they may occur in one or more of the near relations of the patient. Déjérine gives a good example of this associated tendency persisting through six generations of one family. Cases of exophthalmic goitre occurred in four generations while various forms of nervous disease appeared in all six generations. Marie mentions one case in which the father suffered from paralysis agitans, a maternal aunt from insanity, and two children of a sister from epilepsy, and another in which the father was epileptic and a brother neurasthenic. Gowers [123] gives an instance of two sisters who were under his care, one for exophthalmic goitre and the other for epilepsy.

Locality.—Local influences do not appear to take any part in the production of exophthalmic goitre. It is not more common in the regions where endemic goitre is found than in those where ordinary goitre is rarely seen.

Personal Antecedents.—Anæmia is a frequent predisposing cause of exophthalmic goitre. Quinsy and rheumatism not uncommonly precede or accompany the disease. Mackenzie [120] mentions quinsy in 9 and acute rheumatism in 5 out of 40 cases. Rheumatism oc-

curred in 11 per cent. of the 56 cases analyzed by West.[124] Any exhausting process, such as acute illness, loss of blood, sexual excess, pregnancy, parturition, or lactation, may predispose to the disease. Excess in the use of alcohol and syphilis must also be mentioned as being antecedent in some cases. In some instances amenorrhœa, and in some organic heart disease has preceded the onset of the malady, which, however, may develop in a perfectly healthy individual in whom no predisposing influence can betraced.

Exciting Causes.

In many cases of exophthalmic goitre no directly exciting cause can be discovered. In a certain number the first symptoms have so rapidly followed some unusual event that they must be considered as a direct result of the latter. In such cases the exciting cause is generally either some sudden mental or physical shock or else long-continued anxiety or grief sufficient to produce a powerful emotional disturbance. Thus a common cause is a sudden fright which may or may not be accompanied by physical injury. Great fatigue, sexual excess, and cold have also been considered to be the exciting causes in some cases. Mackenzie[120] gives some good examples of events to which some of his patients attributed their illness, such as a severe burn, being thrown out of a trap, a drunken man falling in at the door unexpectedly, terror caused by being in a solitary house. Laycock[125] published a case in which the symptoms rapidly developed after a severe fright, and Trousseau relates the case of a lady in whom enlargement of the thyroid gland, violent palpitation, and exophthalmos developed within a few hours of the receipt of the news of the sudden death of her father. Von Graefe[126] has recorded the case of a man, twenty-two years of age, who succeeded in performing coition after prolonged resistance and a struggle lasting for more than half an hour, during which he became much exhausted; the disease began the next day and developed rapidly. Gowers[123] mentions that many cases occurred in Alsace and Lorraine after the war between Germany and France in 1870. Diseases of the nose or pelvic organs, acting as sources of peripheral irritation, appear in some cases to bring on the disease, though their influence may be rather predisposing than exciting.

Exophthalmic Goitre in Animals.

Only a few instances of the occurrence of exophthalmic goitre in animals have been recorded. Roder has reported one case in a cow in which enlargement of the thyroid gland, palpitation with arterial

pulsation, and well-marked double exophthalmos had been present for four years. Buschan [142] refers to two cases which have occurred in Russia. In one instance a four-year-old horse developed the symptoms after a long gallop. Palpitations with very frequent and strong arterial pulsation, enlargement of the thyroid gland, and progressive weakness were followed by double exophthalmos. Death took place at the end of a month. In the other case the palpitation, goitre, and exophthalmos developed in a bitch seven years old. Under treatment with iodine the symptoms disappeared at the end of three months. Cadiot has also seen a case in a horse that suffered with palpitation, goitre, weakness, and emaciation.

Symptoms.

The symptoms of exophthalmic goitre may conveniently be divided into primary and secondary. Of the former two or more are always present, whereas the number of the latter which may be observed in any one case varies very considerably. The primary symptoms are enlargement of the thyroid gland, increased frequency of the contractions of the heart, and exophthalmos. To these may be added general nervousness and fine tremor, two symptoms which are rarely absent. In most well-marked cases all these five symptoms may be observed associated with some of the numerous secondary manifestations. Of the primary symptoms the exophthalmos may be entirely absent, the enlargement of the thyroid gland may be so slight as to escape notice, though it is rarely absent

FIG. 49.—Exophthalmic Goitre.

altogether, but there is always an increased frequency of the pulse. General nervousness is also an almost constant symptom and the tremor is seldom absent altogether. The appearance of a person

FIG. 50.—Exophthalmic Goitre.

suffering from fully developed exophthalmic goitre is most striking. The prominent eyeballs with the white sclerotic exposed all round the margin of the cornea give a startled expression to the face which, combined with the enlarged thyroid gland, the general unrest of the patient, and the tremor of the hands forms a clinical picture which can be easily recognized, as in Figs. 49 and 50, the latter of which was taken from a patient sent to me by Dr. Craggs, of Newcastle.

ENLARGEMENT OF THE THYROID GLAND.

The thyroid gland is visibly enlarged in the great majority of cases of exophthalmic goitre. In examining the thyroid gland it must be remembered that the normal gland can scarcely be felt and

that it is extremely difficult to ascertain its actual size unless the patient is unusually thin. Thus whenever the gland can be distinctly felt it is almost sure to be enlarged, and in some cases in which no enlargement could be detected during life the gland has been found to be distinctly enlarged after death. The frequency of cases in which no enlargement has been observed is illustrated by the following table:

	Number of cases observed.	Number of cases in which no enlargement was detected during life.
Lewin	27	2
Mackenzie	28	7
Mannheim (own cases)	41	6
" (collected cases)	27	3
Marie	16	5
Maude	15	0
Murray	22	2
Russell Reynolds	49	1
Von Dusch	58	3
	283	29

Thus in twenty-nine of 283 cases no enlargement of the thyroid gland was observed. Maude, who has been able to keep his cases under observation for long periods, has found some enlargement of the thyroid gland in each. If all cases were observed as closely, I believe that some enlargement would be found at one time or another in nearly every one. In a few, as in one of my own in which I could not be certain of the condition of the gland, the thyroid gland may be situated so low down in the neck that a moderate degree of enlargement could not be detected if present. I know of no case of fully developed exophthalmic goitre in which the gland has been proved to be normal in size by a post-mortem examination.

The enlargement of the thyroid gland may be the first symptom to develop and it may even exist for some years before any of the other manifestations arise. The symptoms of exophthalmic goitre sometimes develop in a patient who has had an ordinary parenchymatous goitre for years previously. Sasvènes [127] has described three such cases. In one the goitre had existed for twenty-three years and in another for ten years before the other symptoms developed. Maude [128] has observed seven cases in which the symptoms of exophthalmic goitre have appeared in women who had been goitrous previous to the onset of the disease. In other cases the enlargement occurs simultaneously with the exophthalmos or it does not appear until after the exophthalmos has become quite distinct. The increase in size generally takes place gradually, but occasionally the gland swells up in the course of a few hours. As a rule the enlargement is

moderate in degree and it rarely attains the large size which is sometimes seen in parenchymatous goitre. Frequently the swelling is not sufficient to cause more than a fulness of the front of the neck, though the enlarged gland may then readily be felt by the finger. The enlargement is generally uniform and often symmetrical, but in many cases one lobe is larger than the other. Where this is the case the right lobe is nearly always the larger of the two. The right lobe of the normal thyroid gland is often slightly larger than the left, and where this difference exists naturally it becomes more marked when the gland is enlarged. In rare cases the isthmus only is enlarged. In exceptional cases only one lobe is enlarged and this may be associated with unilateral exophthalmos. In such cases the thyroid gland is usually increased in size on the same side as the exopthalmos, though a case has been recorded by Yeo [129] in which for a time only the left eyeball was prominent, while the enlargement of the thyroid gland was limited to the right lobe, and when exophthalmos developed on the right side the left lobe became enlarged. The swelling is firm and elastic, but its consistence may vary slightly from time to time. The size of the enlarged gland varies. In the earlier stages it gradually increases in size, and it may then become stationary with slight temporary variations. In the later stages of the disease the gland not infrequently grows harder owing to the formation of fibrous tissue within it. This may gradually contract, cause a diminution in the size of the goitre and end in recovery. Indeed this process may continue until it destroys the actual gland substance and the exophthalmic goitre is then followed by myxœdema. The enlarged gland is very vascular and under the influence of emotion it may enlarge slightly from an increase in the amount of blood passing through it. The gland often pulsates, but it is difficult to distinguish between pulsations transmitted from the carotid arteries and true pulsation of the gland itself due to sudden expansion caused by the fresh blood driven into it at each systole of the heart. The veins over the gland are sometimes visibly dilated. A thrill can often be felt when the finger is laid over the gland, and a blowing murmur can be heard with the stethoscope in some cases.

Circulatory System.

Heart.—A marked increase in the force and frequency of the contractions of the heart is the most constant of all the symptoms of exophthalmic goitre. Indeed nearly all observers consider the presence of some acceleration of the pulse as an essential feature of the disease, and Charcot maintained that it was present in all cases. The

acceleration of the pulse is nearly always the first symptom to develop except in those cases in which the phenomena become engrafted upon those of a pre-existing goitre. The onset of the acceleration may be rapid or it may be gradual and intermittent, the pulse returning to its normal rate between the attacks of palpitation. The action of the heart is very similar to that which occurs as the result of violent exertion or intense emotion. Nearly all patients are conscious of the palpitation, especially when it is temporarily increased by any excitement. It is, however, not so distressing as a sudden attack of ordinary palpitation is to a healthy person. The actual rate of the pulse varies much in different cases. In some the increase is slight and the pulse does not beat more than 90 or 100 a minute. In fully developed cases the rate is generally higher and frequently ranges from 120 to 150; occasionally it may reach 180 or even 200. The heart is very irritable and a slight exertion or trifling emotional disturbance readily increases the rate of the pulse by twenty or thirty beats a minute. The pulse is small, soft, and regular, and in some cases distinctly dicrotic. Irregularity of the pulse may arise from cardiac disease occurring as a complication. The blood pressure in the radial artery has been measured by Marie in two cases and in each instance was found to be normal, and there is no evidence to show that it is diminished. On inspecting the chest the cardiac impulse is seen to be increased in force and to extend over a larger area than usual. There is often strong epigastric pulsation. The cardiac impulse feels short, sharp, and sudden. When the disease has lasted for some time the area of dulness may be enlarged to the left owing to dilatation of the left ventricle. The heart sounds are louder than usual and in some cases they are audible to the patient; Graves described a case in which he could hear them at a distance of four feet from the chest. In early uncomplicated cases the sounds are generally clear and no murmur is heard; but sometimes they are rough and blurred, and murmurs are by no means uncommon, especially in cases of some duration, though observers differ as to their actual frequency. Russell Reynolds [130] heard a murmur in two-thirds of his cases, Mannheim [116] in five out of forty-one cases. A systolic murmur may be heard all over the cardiac area, more loudly over the base than over the apex. Such a murmur Gowers [123] believes to be produced at the orifices of the large vessels. When anæmia is present the murmur is hæmic in origin. In some cases the murmur has all the characters of that produced by disease of the mitral or tricuspid valves. In these cases the murmur is caused by mitral or tricuspid regurgitation which occurs as a result of incompetence of the valves from dilatation of either the right or the left ventricle. Occa-

sionally endocarditis may occur as a complication, and various valvular lesions may develop, each with the usual attendant murmur.

Arteries.—The arteries pulsate forcibly, partly owing to the increased force of the contractions of the heart and partly owing to dilatation from the loss of their natural tone. The pulsation is seen and felt most distinctly in the carotid arteries. Strong pulsation may also be observed in the abdominal aorta and femoral arteries. Kahler records a case in which the patient suffered greatly from the abdominal pulsations which could be relieved only by keeping the thighs constantly flexed on the abdomen. Gerhardt has seen pulsation of the spleen in one case of three months' duration. Some patients complain that the whole body seems to throb. Gowers [123] mentions that pulsation can sometimes be observed even in the arteries of the retina. A loud systolic arterial murmur can be heard in the vessels of the neck and occasionally in those of other parts of the body as well.

Ocular Symptoms.

Exophthalmos.—The prominence of the eyeballs which is such a conspicuous feature in many cases of exophthalmic goitre is not present in all. It is rarely the first symptom to appear, but generally develops later than the tachycardia. Exophthalmos is present in some four-fifths of the cases. Mannheim [116] mentions that it was present in 74.1 per cent. of 112 cases collected from recent literature, in 85.3 per cent. of 41 cases of his own, and in 81.5 per cent. of 27 cases published by Lewin. The onset of the exophthalmos is generally gradual. It may be rapid, becoming quite distinct in the course of a few days in exceptional cases. Both eyeballs are equally affected in most cases, but sometimes the prominence appears first on one side only and remains more marked on that side than on the other. In such cases the right eyeball is most frequently the first affected and remains more prominent than the left. In rare cases one eyeball only is prominent. When this occurs it is on the same side as the larger lobe of the thyroid, an exception to this rule being the remarkable case, reported by Yeo, to which reference has already been made (p. 773). The degree of prominence of the eyeballs varies greatly in different cases. In some it is so slight as scarcely to attract attention. In a well-developed case the eyes have a staring and startled expression and the white sclerotic shows clearly all round the edge of the cornea. Sometimes the eyeballs are so prominent that the insertions of the recti muscles are visible and the eyelids are unable to meet over the front of the eye. It is said that the eyeball has actually been dislocated from the orbit. The exophthalmos

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may vary in degree from time to time in the same patient, being more marked when the action of the heart is increased. In some cases it appears only under special circumstances, the eyeballs being normal in appearance during the intervening periods. Thus in one case the exophthalmos appeared only during choreic attacks (de Mussy) and in another only with the mental disturbance which accompanied menstruation (Savage). The prominence of the eyeball causes it to appear to be enlarged, and Neumann has estimated that the diameter is actually increased by one-tenth. The exposure of the cornea may lead to certain secondary changes. The cornea may become dry or there may be superficial ulceration or conjunctivitis. Inflammation of the cornea may even lead to sloughing. In one case recorded by Craig the lower portion of each cornea was ulcerated. There was no anterior chamber and the patient could only distinguish light from darkness. The prominence of the eyeballs has been attributed to various causes, such as excessive deposit of fat in the orbit, overfilling of the blood-vessels, and spasm of the small muscular fibres known as Müller's muscle.

V. Graefe's Symptom.—This symptom consists of a defective descent of the upper eyelid when the eyeballs are directed downward. In health, as the eyes follow an object descending from a level above their own to one below it, the upper eyelids descend simultaneously so that though the eyeballs are rotated downward none of the white sclerotic above the cornea comes into view. When von Graefe's symptom is well marked the upper eyelids remain in their previously elevated position and do not follow the downward movements of the eyeballs, so that as the gaze is directed further and further downwards more and more of the white sclerotic becomes visible above the upper border of the cornea. If the patient then looks upward again the eyeballs alone turn upward until they come into their normal relationship with the upper eyelids. If the eyes are then directed still further upward the eyeballs and upper eyelids move together as in health. In some cases the descent of the eyelid is only delayed or takes place in a jerky or irregular manner. In such cases the upward movement of both eyes and eyelids is performed in the normal manner. When the eyes are closed as in sleep the upper eyelid descends in the usual way. A peculiar modification of this symptom has been observed by Ramsay [131] in which the upper eyelid followed the first part of the downward movement of the eyes, then remained stationary while the eyes looked still further downward, till after a few seconds a spasmodic retraction of the lid took place which brought the white sclerotic into view. Von Graefe's symptom is present in a good many cases of exophthalmic goitre. Mannheim [116]

found it present in 18 out of 41 cases, but Russell Reynolds [130] in 4 only out of 49. It may be associated with but a slight degree of exophthalmos, or be altogether absent when exophthalmos is well marked, while it occasionally occurs in persons who are not suffering from exophthalmic goitre at all. Sharkey observed it 14 times in 613 patients, none of whom had exophthalmic goitre. Von Graefe considered that this symptom was due to the spasmodic contraction of the involuntary muscular fibres of Müller which aid the levator palpebræ superioris in raising the eyelid and which receive their nerve supply from the cervical sympathetic.

Stellwag's Sign.—When this is present the palpebral fissure is increased in width owing to the persistent retraction of the upper eyelid. This is due to an increase in the natural tonic contraction of the elevator of the upper lid which is always in action while the eyes are open. This spasm is possibly due (as Maude suggests) to paresis of the opposing muscle, the orbicularis. The eyelid is thus maintained in a more elevated position than usual. When present it increases the staring expression already produced by the exophthalmos. It may occur either with or without v. Graefe's symptom. Thus Mannheim found among 41 cases of exophthalmic goitre that 10 exhibited both symptoms, 6 showed v. Graefe's symptom without Stellwag's, and 4 Stellwag's without Graefe's. It varies in degree from time to time and becomes more marked under the influence of emotion. It is rarely unilateral. Retraction of the lower eyelid has seldom been observed, though Griffith has described it in 3 cases. Möbius [132] considers that Graefe's symptom occurs as a result of Stellwag's and that the defective descent of the upper lid is due to the elevator spasm which has to be overcome before the lid can descend, causing the lid to lag behind when the patient looks downward. If this be so, it is difficult to explain how one symptom may occur without the other.

Defective Convergence.—In 1883 Möbius [132] first drew attention to a deficiency in the power of convergence of the eyes which is present in a certain number of cases of exophthalmic goitre. This deficiency may be demonstrated by causing the patient to watch the finger as it is brought gradually nearer and nearer to the eyes. At first the eyes converge naturally, but when a certain distance from them is reached, a distance which varies in different cases, and in the same case at different times, the eyes suddenly become parallel and only the one which is turned inward continues to be directed toward the finger. This symptom occurs independently of any ocular paralysis. The patient is unaware of it and there is no double vision.

Ocular Paralysis.—Various forms and degrees of paralysis of the

external muscles of the eye have been observed in exophthalmic goitre. A general weakness of all the muscles of the eye, which is present in some cases, may be due rather to mechanical stretching of the muscles as a result of the exophthalmos than to any nervous defect. The paralysis may involve all the ocular muscles or only one, it may be complete or partial, and it may be bilateral, unilateral, or more marked on one side than on the other. Complete ophthalmoplegia externa may occur alone or in association with paralysis of other nerves. Bristowe [133] has recorded a case in which, in addition to ophthalmoplegia externa, there were hemiplegia, hemianæsthesia, which affected the special senses as well, bleeding from the ears, and fever. In Ballet's case the paralysis of the ocular muscles was associated with paresis of the soft palate, tongue, and facial muscles. In Warner's case there was also paresis of the facial and trigeminal nerves. Maude [134] relates a case in which paresis of the external rectus, and probably of the superior oblique, was preceded by facial paralysis and was quickly followed by general ophthalmoplegia. This sequence he concludes was due to some lesion attacking in turn the nuclei of the seventh, the sixth, the fourth, and the third nerves. Paralysis of a single ocular muscle is uncommon. Liebrecht observed an instance of paralysis of the right external rectus muscle and Mackenzie one of paresis of the external recti, causing double vision when the eyes were strongly directed to one side or the other. Finlayson has recorded an example of paralysis of the third nerve only, and Féréol one in which the fourth nerve alone was affected. The paralysis of the third nerve is seldom complete and even in complete external ophthalmoplegia the levator palpebræ superioris generally escapes. Ptosis may, however, occur without ocular paralysis, as in a case recorded by West and in three cases mentioned by Mannheim.[116]

The eyelids are occasionally œdematous, and Gowers [123] has seen œdema of both eyelids and conjunctiva associated with only a slight degree of exophthalmos. A tremor of the eyelids may often be observed when the eyes are shut, a symptom which, however, frequently occurs in those who are suffering from some nervous disease. Nystagmus has occurred occasionally, though it has generally been considered to be hysterical in origin. The pupils are usually equal, of medium size or somewhat dilated. They react normally to light and to accommodation. The vision is rarely affected by the prominence of the eyeballs; Kart and Wildbrandt found diminution of the field of vision in twenty cases. This occurred also in two of Charcot's cases who had hysteria, however, to which he attributed the symptom. The fundus of the eye is generally quite normal in ap-

pearance, though Gowers has seen pulsation of the retinal arteries and in one case œdema of the disc. Emmert has seen atrophy of the optic nerve.

Nervous System.

A considerable number of nervous symptoms are liable to occur in exophthalmic goitre. Some of these occur frequently, others are rarely seen. For the most part these symptoms indicate a functional rather than a structural derangement of the nerve centres, as they are often temporary in duration and variable in intensity. The nervous symptoms which occur in connection with the movements of the eyeballs and eyelids have been already described and so require no further consideration. Two nervous symptoms, tremor and general nervousness, are so frequently present that they are usually considered as primary symptoms.

Tremor.—In the great majority of cases, and possibly in all, tremor is present at one period or another. It occasionally comes on early, but more frequently it does not appear until other symptoms have been present for some time. The character of the tremor may most conveniently be observed when the hand is held out with the palm directed downwards. When the tremor is slight and cannot easily be seen, it may readily be felt by laying the palm of the hand lightly upon the back of the patient's fingers when the latter's hand is held out. The tremor affects the flexor and extensor muscles of the wrist and not the intrinsic muscles of the hand, so that the fingers do not vibrate independently but the hand moves as a whole. The tremor is constant in its character while it lasts. The rhythm is rapid and regular. Marie found from tracings taken in eleven cases of exophthalmic goitre that the movements occurred from eight to nine and a half times a second, the average rate being eight and a half a second. The amplitude of the tremor is small but variable. The changes in it, which are much more readily appreciated when recorded by a tracing than when watched or felt, occur independently of the cardiac or respiratory rhythm and their cause is not known. If the foot is held out it will be seen to tremble in the same manner as the hand. In some cases the whole body appears to tremble, and the tremor can be distinctly felt by laying the hand upon the head, trunk, or limbs. The tremor is generally equal on the two sides of the body, but it has been observed to be unilateral when the goitre and exophthalmos were also confined to one side; it may even be confined to one limb, or it may be more marked in one limb than in the others; it is more distinct when a patient is sitting than when she is lying down; it is increased by excitement and by attempts to execute any movement

with unusual care. As a rule the tremor does not impede the movements of the hands, but when excessive it may hinder sewing or writing, and in rare cases it may be so great in the legs as to prevent walking. The tremor of exophthalmic goitre is like that which is developed in the monkey after removal of the thyroid gland, having the same rate of oscillation. It thus differs from the tremor of old age in which there are four and a half to five and a half vibrations each second, and from that of paralysis agitans in which the rate is variable, or of disseminated sclerosis in which the rate is from three to six per second. The tremor of general paralysis and of alcoholism is also rapid (eight to nine per second), but it is much less regular in amplitude and the individual fingers tremble. The tremors of fatigue, and of weakness, as during convalescence from acute illness, closely resemble that of exophthalmic goitre.

Mental Condition and Insanity.—In almost all cases of exophthalmic goitre there is a peculiar mental condition which is so constant that it may be considered as one of the primary symptoms of the disease. The change from the previous normal condition may be only slight, but it is generally quite sufficiently well marked to attract the attention of the friends of the patient. This mental condition may be abnormal only at times; it is very variable and may occur in all degrees of severity, amounting in some cases to complete insanity. Most commonly there is a condition of general nervousness in which very trivial circumstances are sufficient to make the patient excited and apprehensive. This peculiar condition of nervous expectancy is not unlike that of a girl just going to her first ball or of a young man just going up for a *viva-voce* examination. There is often restlessness of body and irritability of temper or intolerance of contradiction. Much unnecessary care and trouble are often expended over some trifling object which is quite unworthy of so much attention, and yet there is in many cases a great want of true application so that no single occupation is followed for any length of time. Patients are often bright and hopeful about their recovery, though this state of mind may alternate with fits of depression. Some are nearly always in low spirits and shed tears on the slightest provocation. Occasionally the moral nature appears to be changed and the patient becomes morose, quarrelsome, and untruthful. Sleep is often disturbed and the memory becomes defective. The mental symptoms may increase in severity until the patient becomes insane. Actual insanity should perhaps be considered as a complication rather than as a symptom, though the simultaneous improvement in the mental and bodily condition in some cases indicates that the former may occur as a direct result of the latter. Melancholia and mania are the most common types

of insanity, though recurrent mania and general paralysis also occur. Savage [135] has observed three cases in each of which there was a "restless condition of mental irritability and violence," practically an exaggeration of the mental condition which so often occurs without any insanity. Out of 44 cases collected by Martin and by Schenk, which are tabulated by Mannheim, mania occurred in 16, melancholia in 10 (in 2 of which there was excitation and in 1 ideas of persecution), delusions of persecution in 10, "insanity" (*wahnsinn*) in 3, excitation in 2, delirium in 1, exaltation in 1. Of these cases 12 died, 6 recovered, 5 improved, 4 apparently improved, 4 did not improve, and of the other cases no account of the progress is given. When insanity occurs it is often the cause of death.

Other nervous symptoms are not so constant as those we have just dealt with and can only be regarded as secondary or occasional symptoms. A troublesome symptom which occurs not infrequently is a sudden giving way of the legs, so that the patient at once falls to the ground without any previous feeling of faintness or giddiness. This has been observed by Mackenzie [120] in 12 cases. Charcot has drawn attention to a marked weakness of the legs or paraparesis which appears to be preceded by the giving way of the legs. When paraparesis is present the muscles are weak and flaccid and the knee-jerk and superficial reflexes may be diminished or absent. In this condition sensation is unaffected and the electrical reactions are not altered. The loss of power is temporary only and it appears to be functional; it occurs without symptoms of hysteria, and so is a symptom and not a complication of exophthalmic goitre. Complete paraplegia and hemiplegia have been observed in a few severe cases. Painful cramps occur in some cases. Mackenzie found this symptom in thirteen out of fifteen cases. These cramps generally occur at night; the feet and legs are most frequently attacked, though the hands alone are affected in some. Localized muscular atrophy is a rare symptom. The muscles of the extremities appear to be the most liable to be affected. Astasia-abasia, or inability to stand or walk while the legs can be freely moved when lying down, has been observed in several cases by Maude. Many other symptoms denoting functional derangement of the nervous system have been observed in those suffering from exophthalmic goitre, but as they are to be regarded as complications rather than symptoms they will be considered separately.

Cutaneous System.

Pigmentation.—A dark coloration of the skin of persons suffering from exophthalmic goitre has been described by several observers.

Begbie in 1862 noticed the dark color of the skin of the face of one of his patients. More recently Drummond [136] has described this symptom in six cases. Other cases have been recorded in which more or less marked bronzing of the skin had taken place. This pigmentation is most pronounced and most frequently seen in those situations in which pigment is naturally most abundant in the skin. Thus the skin round the eyeballs is often darker than natural. The skin of the face may be uniformly bronzed to a slight degree, or the coloration may occur in dark-brown patches upon the cheeks. The neck, the axillæ, the areolæ of the nipples, the genital organs, the upper part of the inner surface of the thighs, and the flexures of the joints are most frequently affected. Any part of the skin which is subjected to constant pressure by the clothes is also liable to become dark in color, as, for example, just below the knee where the garter is worn, or where the body is compressed by the corset. In one of my own cases the pigmentation was very well marked, but it was chiefly limited to the exposed portions of the face, hands, and arms, being much less distinct where the skin was covered by clothing, the appearance of the patient being thus very similar to that of a woman who has been working in the fields under a summer sun with her sleeves rolled up. The color of the skin may vary from a pale liver color to a dark mahogany brown. The pigmentation may occur in sharply defined patches, as in Drummond's case, or it may gradually shade off at the edges or be diffused over a considerable area. The pigmentation may be as distinct as it is in the early stages of Addison's disease, but it is never so great as it may be in an advanced case of that disease. The brown patches which occur so frequently in the mucous membrane of the mouth in Addison's disease are not seen in cases of exophthalmic goitre. Two cases of exophthalmic goitre in which the patches were observed by H. Oppenheim and by A. Eulenburg were probably suffering from Addison's disease at the same time. The pigmentation fades away as the other symptoms subside and the general condition of the patient improves. Patches of leucoderma are present in some cases, but this change in the skin appears to be less frequent than pigmentation. Maude has observed small oval patches of light-colored skin, "reversed freckles," on the pigmented eyelids.

Rashes.—Various rashes may occur in exophthalmic goitre. Erythema has been noticed by Sidl and by Marie. A double form of rash, which has been described by Joffroy [137] and by Maude, consists of dusky papules like the eruption of measles in its later stages, and purpuric spots. Urticaria also may appear.

Excessive Sweating.—There is frequently an increase in the secretory activity of the sweat glands in exophthalmic goitre. In some

cases the skin as a whole is much more moist than usual, the skin of the face especially being greasy. Profuse general or localized sweatings may occur, especially in acute cases. The sweating may take place several times in a day and last for an hour or more at a time. The skin often feels warm, and patients may complain of sensations of heat or of hot flushings. The flushings may affect some particular part of the body, as the back, or they may occur only on one side, or may be general.

Diminished Electrical Resistance.—It was first pointed out by Vigouroux that in many cases of exophthalmic goitre the natural electrical resistance of the skin is much less than usual. Wolfenden [138] found that, while in health the average resistance of the skin to a moderate galvanic current amounts to four or five thousand ohms, in eight cases of exophthalmic goitre in which the same current was employed the resistance was only from five to seven hundred ohms. In two of the eight cases the resistance was only two hundred and three hundred ohms respectively. This symptom has also been investigated by Charcot, Martins, Eulenburg, Kahler, and Cardew, and their observations show that in a large number of cases of exophthalmic goitre there is certainly a marked diminution in the electrical resistance of the skin. This symptom, however, is not present in all cases, and it occurs occasionally in health as well as in some other diseases. It is apparently a direct result of the moist condition of the skin, which is due to the dilatation of the capillaries and the excessive activity of the sweat glands, which may be sufficient to moisten the skin without producing obvious sweating. The more moist the skin becomes the better it conducts, so that the resistance appears to vary according to the amount of moisture present in the skin. Thus Cardew [139] has shown that in other diseases the electrical resistance is diminished as soon as the skin begins to perspire.

Œdema.—Œdematous swelling of the skin in various parts of the body occurs frequently in exophthalmic goitre, and it is important to discriminate between œdema which may be considered as a direct result of the primary disease and œdema which occurs as a result of some complication. Thus in some cases of exophthalmic goitre œdema of the feet and legs occurs as a result of anæmia, organic heart disease, varicose veins, cardiac weakness, or renal disease. Œdema which occurs as a symptom of one of these complications does not, however, concern us further here, as it is not a direct symptom of the original disease. But, apart from such complications, œdema does occur in exophthalmic goitre apparently as a direct result of the disease itself. Not infrequently there is some swelling of the feet and especially about the ankles. Such œdema has been ascribed by

Maude and others to vaso-motor paralysis. A somewhat similar œdema, probably also of nervous origin, occurs in irregular patches of transitory existence on various parts of the face, neck, arms, and hands. In connection with this may also be mentioned the supra-clavicular swellings resembling those which occur in myxœdema and cretinism, which were first described by Rendu. In a few cases the presence of a general solid œdema like that which is present in myx-œdema has been observed and has led to the conclusion that the two diseases may coexist; but this is open to considerable doubt. It may here be mentioned that a case, which had been published as an example of combined exophthalmic goitre and myxœdema, was subsequently, by the advice of the writer, treated for several months with thyroid extract, but little or no diminution of the swelling took place. This indicates that the swelling in this case did not depend upon loss of thyroid secretion and so was not myxœdematous. A swelling of the eyelids which is seen in some cases is considered by Vigouroux to be due not to œdema, but to paresis of the orbicularis muscle, as it disappears when electricity is applied to that muscle. Möbius [132] has described a remarkable case with cardiac weakness and emaciation in which there was œdema of the lower half of the body. The œdema was only slight in the feet, increased from below upwards, was greatest in the abdomen, but ceased at the level of the umbilicus. The peritoneum, however, was free from fluid.

Hair.—The nutrition of the hair is generally more or less affected in exophthalmic goitre. This often leads to loss of hair from the scalp, axillæ, and pubes. In a few cases actual baldness may occur in some situations as a result of atrophy of the hair. Mannheim found that loss of hair had occurred in fifteen out of forty-one cases. Bryson records a case in which all the hair was lost with the exception of the eyebrows and an oval patch on the crown of the head. In one of my cases the head was quite bald, the eyebrows and eyelashes had also been lost, and some but not all of the pubic hair had come out. Sir T. Grainger Stewart and Dr. G. A. Gibson [140] draw attention to the fact that when some hair is lost that which remains becomes profoundly changed. They mention one case in which this change in the nutrition of the hair was much greater on one side of the body than on the other.

Nails.—Marked changes in the condition of the nails have been described by Sir T. Grainger Stewart and Dr. G. A. Gibson.[140] The nails in some cases become thin and brittle, and occasionally they have a corrugated appearance with an opacity of a yellowish color. Both hands may be equally affected or one more than the other, and individual nails may show more marked changes than the rest.

Digestive System.

Gastro-intestinal disturbances are of frequent occurrence, and they generally take the form of attacks of diarrhœa of a somewhat peculiar type. Diarrhœa was noted as a symptom in forty-six out of one hundred and fifty-eight cases collected by Mannheim[116] from various sources. The diarrhœa comes on suddenly without any apparent cause. As a rule there is little or no pain. The bowels may act two or three times in a day, and then the attack may be at an end, or it may be prolonged for two or three days, after which it ceases as suddenly as it began. The motions are copious, loose, and light in color. The tongue remains clean. If the attack is slight little general disturbance is caused by it. In severe attacks the bowels may act as many as fifteen to eighteen times in the twenty-four hours. Vomiting may occur and in rare cases there may be hæmatemesis and melæna. If the attack is severe or prolonged for several days exhaustion and emaciation occur as a natural result, and the attack may even end fatally. The diarrhœa may be accompanied by a rise of temperature and so may simulate an attack of typhoid fever. These attacks may be accompanied by a voracious appetite, which may last for a short time only or all day. The increase of appetite may, however, be combined with considerable and progressive emaciation. The diarrhœa in the great majority of cases appears to be of nervous origin, and it has been suggested by Maude that it is produced by an increase in the intestinal secretions comparable to the sweating of the skin. Increased peristaltic action of nervous origin also helps to produce this symptom. It must, however, be remembered that Grainger Stewart and Gibson have found extensive catarrh of the alimentary canal in two fatal cases of exophthalmic goitre.

Respiratory System.

An increase in the frequency of respiration is often a noticeable symptom. In some cases it occurs as a result of cardiac weakness or anæmia and in others as a result of the pressure exercised by an enlarged thyroid gland upon the trachea. Apart from these causes, however, rapid breathing of nervous origin is not uncommon. The respirations may be accelerated to 30 or even 38 in a minute. The paroxysms of coughing which occur in some cases without any affection of the respiratory organs are also of nervous origin. The dyspnœa may be accompanied by cyanosis of the face and swelling of the vessels in the neck, and fatal asphyxia has occurred in two cases

recorded by Shingleton Smith and by Bristowe. In two cases recorded by Maude and by Bramwell pulmonary œdema with profuse bronchial secretion and expectoration of blood-stained sputum occurred. The profuse bronchial secretion is considered by Bramwell to be due to vaso-motor paralysis and to be comparable to the sweating and to the diarrhœa from increased intestinal secretion. Bryson considers that the expansion of the chest with inspiration is considerably diminished, and maintains that the less is the inspiratory expansion of the chest the worse is the prognosis. Mannheim, who has made careful observations upon this point, has, however, been unable to confirm Bryson's conclusions.

Urinary System.

Polyuria is not infrequent. The over-activity of the kidney is comparable to that of the sweat glands or of the intestinal mucous membrane, which, as we have already seen, is common in this disease. Albuminuria was described in several cases by W. Begbie. It is generally intermittent and the urine is free from casts. In some cases it occurs in conjunction with dilatation of the heart (Grainger Stewart and G. A. Gibson). In the great majority of cases the albuminuria is undoubtedly functional in origin and does not indicate any actual disease of the kidney. Glycosuria has been observed by many writers.

Generative System.

Opinions differ as to the effect of the disease upon menstruation. Thus Russell Reynolds [130] found menstruation normal in the great majority of cases (forty-six out of forty-nine), while others have drawn attention to the frequency of amenorrhœa. Pregnancy does not often occur, but it may do so and be accompanied by improvement in the patient's condition (Trousseau, Charcot). In several cases a defective development of the uterus, ovaries, and mammary glands has been noticed (Kleinwächter, Cheadle, Maude). Loss of sexual desire and impotence in the male has several times been recorded.

General Symptoms.

Sensations of heat are often experienced but are not necessarily an indication of a rise of temperature. Mackenzie has found no rise of temperature above the normal level in patients who complained of feeling hot. H. Bertoye [141] has made very careful observations on the temperature in exophthalmic goitre. He found that a rise of

temperature often took place. The febrile attacks may be either transitory or persistent. If the fever persists it may be of either intermittent or remittent type. In one patient, who suffered from amenorrhœa, severe febrile attacks occurred at the times when menstruation should have taken place. In some cases during the febrile attack the face is flushed, the tongue is coated, the head aches, and other common symptoms of pain are present. In other cases the rise of temperature is unaccompanied by any marked constitutional disturbance. Anæmia often occurs in the course of exophthalmic goitre; Russell Reynolds[130] found it present in forty-eight out of forty-nine cases. It is, however, by no means a constant symptom, as in many cases the amount of hæmoglobin and the numbers of red and of white corpuscles in the blood are within the limits of health.

Rapid emaciation is a marked feature in some cases. Mannheim mentions one case in which four pounds were lost in eight days, another in which forty pounds were lost in a year, and a third whose weight fell from one hundred and eighty-seven pounds to ninety-four pounds in ten months.

Complications.

Epilepsy.—Epileptic attacks may occur in a patient suffering from exophthalmic goitre, but, as Ballet points out, we should distinguish between attacks which occur as a coincidence and those which occur as a complication. Independent epilepsy may develop either before, during, or after an attack of exophthalmic goitre. In some cases the epileptic attacks seem to occur as a direct result of the exophthalmic goitre, and they have been attributed to changes in the cerebral circulation dependent upon the deranged action of the heart.

Hysteria.—Hysteria is a very common complication, occurring in from twenty-five (Reynolds) to thirty-four (Mannheim) per cent. of the cases of exophthalmic goitre. Many of the convulsive attacks which occur in the course of exophthalmic goitre are hysterical in origin. The most frequent signs of hysteria are alterations in sensation and paresis, or actual paralysis, of one or more limbs or groups of muscles. Almost any of the numerous symptoms of hysteria may, however, arise. Thus there may be localized or general convulsive seizures of epileptic, choreic, or tetanic type, more or less distinct paresis, paraplegia, monoplegia, or hemiplegia. Anæsthesia, paræsthesia, hyperæsthesia, and neuralgia are common. Astasia-abasia (Eulenburg, Maude) and contraction of the field of vision (Kast, Wildbrand) have also been observed.

Locomotor Ataxy.—The coexistence of exophthalmic goitre and locomotor ataxy in the same patient has been described in twelve or more cases. Joffroy has observed seven examples of this combination. A few years ago he was kind enough to show me one of his cases at the Salpêtrière in Paris, and at that time he mentioned that there were no less than five patients suffering from combined exophthalmic goitre and locomotor ataxy in that institution.

Rarer Complications.—Paralysis agitans (Möbius), chorea (Gauthier), tetany (Dreyfus-Brisac), Thomsen's disease (Raymond), gangrene of the extremities (Fournier, Olivier, and others), arthritis (Spender, Maude), and osteomalacia (Koeppen) have been observed as complications in rare instances.

Natural Course, Duration, and Termination.

The natural course of exophthalmic goitre is by no means constant, and varies considerably in different cases. In the great majority the course is chronic, and the disease may last for years with occasional variations in severity.

Complete recovery takes place in some cases, but we have not sufficient data to enable us to determine in what proportion of cases this occurs. From the relatively few recorded examples of complete recovery it appears that this does not take place as frequently as was believed by Charcot and by Fagge, who both considered that recovery was common. Gowers mentions a case, in which the symptoms disappeared and had not recurred twenty years after, but had been succeeded by those of myxœdema. Similar cases will be mentioned in considering the pathology of the disease. In some cases the recovery is followed by one or more relapses, the patient being quite free from the symptoms in the intervals.

An acute course, following acute onset, is rare, but such cases, when they do occur, may recover rapidly. Thus Mackenzie [120] quotes a case recorded by Moore in which the symptoms, which developed on hearing of the death of a brother, lasted only two days. In a case published by Solbrig [143] the symptoms entirely disappeared after twelve days. In two others given by Fletcher, an acute onset in which there was complete recovery took place in a month or two. One case has been recorded by Müller [144] in which a girl, aged ten years and a half, recovered six weeks after the onset. Corning [145] also states that the symptoms may develop in forty-eight hours and recovery take place in a few weeks. But these acute cases may also run a rapid downward course and end fatally. In Chevalier's [146] case, within four days the symptoms developed with epileptic convulsions

and ophthalmoplegia externa, followed by death in convulsions. Mannheim,[116] who mentions this case, also mentions Lloyd's case in which the symptoms developed very rapidly and death occurred in three days. Müller[147] has also reported four cases, in three of which the disease developed and ended fatally in from six weeks to three months and a half. Bamberger and Michell Clark have each reported a case with fatal termination occurring within six weeks after the onset of the symptoms.

The great majority of cases, however, run a more chronic course and the symptoms may persist more or less for years. Four or five years is not an uncommon period for the disease to last, and it may continue for as much as twenty years. I have seen one case twelve, another thirteen, and a third twenty years after the commencement of the disease. In the two former the symptoms were well marked. In the last case great improvement had taken place after the thyroid gland had been reduced in size by painting the skin over it with iodine, and at the time I saw the patient the gland appeared to be scarcely larger than normal; there was still a very slight degree of exophthalmos with slight retraction of the upper lids, and the pulse was 92. In this case recovery, though not quite complete, was so far advanced that the patient herself was no longer conscious of the symptoms. This amount of recovery no doubt occurs in many chronic cases, and in such it is only after careful examination that any signs can be found which show that the patient has had exophthalmic goitre. Most of these chronic cases tend to improve, and though recovery may not be complete the improvement is sufficient to enable the patient to live comfortably and enjoy life again. Other chronic cases may go on for years and then finally become worse and die.

Death most frequently occurs from failure of the heart. In these cases it is often preceded by severe attacks of palpitation and breathlessness, œdema of the lower extremities, and marked emaciation. Sudden death, probably from syncope, has been recorded in several cases, notably in one observed by Hale White in which the patient fell back dead when the electric current was applied. With these cases must also be mentioned those in which death has occurred during or shortly after removal of part of the enlarged thyroid gland, though it is not as yet evident whether death in such cases is due to the operation itself or to the anæsthetic. Death may occur from general weakness aggravated by diarrhœa, vomiting, and emaciation. A considerable number of deaths are caused by attacks of intercurrent diseases, the resistance of the patient being diminished by the pre-existing exophthalmic goitre.

Pathological Anatomy.

The Thyroid Gland.

In spite of the large number of papers which have been written on exophthalmic goitre, comparatively few writers have described the changes which are to be found in the thyroid gland. This is the more remarkable when we consider that the thyroid gland is obviously the organ which undergoes more change than any other in this disease. By several the goitre is considered to be a vascular one and the enlargement to be mainly due to increased vascularity, dilatation of the vessels, and hyperæmia. The degree of enlargement of the gland varies in different cases, but it is frequently greater than it appeared to be before death. This is important, for it shows that the gland may be increased in size, even when no enlargement can be detected on palpation of the front of the neck during life. The veins on the surface of the gland are enlarged and their walls are thin. The cut surface of the gland is fairly uniform in appearance. It is paler in color and less colloid material exudes from it than in health. With the exception of the veins already mentioned, no marked increase in the vascularity of the gland is visible to the naked eye. There may or may not be some thin-walled cysts.

Microscopical Appearances.—The microscopical appearances of the thyroid gland indicate that the most important change is a general increase in the amount of the secreting gland tissue. The increase in the size of the gland is seen to be due to a great increase in the amount of glandular tissue. This increase may be fairly compared to that which takes place in the mammary gland during lactation. The change is a general one, but is not a simple hypertrophy, for the new tissue differs considerably in appearance from that of the normal thyroid gland. The number of acini is increased very considerably, while the normal cubical epithelium is replaced by one of a columnar type (Greenfield[148]). There is active secretion of colloid material which, instead of being stored up in the alveoli of the gland, is rapidly absorbed. Thus, in sections of the gland, less colloid is seen than in health, and it is replaced by a more mucinous fluid. In some of the alveoli catarrhal changes with desquamation of the epithelium are not infrequently to be observed. The epithelial cells which have been recently shed are to be seen lying in the alveoli. Greenfield[148] has observed that a great number of newly formed tubular spaces are produced which are lined by cubical epithelium. These closely resemble the tubules of a secretory gland. In some cases there is a distinct increase in the amount of connective-tissue

stroma. In old-standing cases there may be very marked chronic interstitial thyroiditis leading to atrophy of the glandular substance. This change may, indeed, lead to so much atrophy that myxœdema develops as a result of the thyroid inadequacy.

Blood-Vessels.—Both arteries and veins are fairly abundant. The vascular supply, however, is not greater than we should expect to find in a gland which has all the appearances of having undergone active evolution. The arteries may be actually increased in size and irregularly dilated, but, as Greenfield [148] points out, a similar enlargement takes place in the uterine and mammary arteries when the organs are in full functional activity.

The Thymus Gland.

The thymus gland is often persistent, and, as Möbius [132] and others have found, it may be considerably enlarged. The number of observations upon the condition of the thymus is not as yet sufficiently great to enable any conclusions to be drawn as to the frequency of the enlargement of this gland.

Nervous System.

Acting upon the supposition that exophthalmic goitre is essentially a disease of the nervous system, many observers have made very thorough examinations of all parts of the nervous system in this disease. The main result has been negative, for no one constant lesion has been found which seems adequate to explain the symptoms of the disease.

Brain.—In the brain no morbid change has been observed in the majority of cases. In one case, in which there was hyperæmia of the brain substance and also of its membranes, maniacal symptoms had occurred before death.

Medulla Oblongata.—Definite lesions have been found in the medulla more frequently than in any other part of the nervous system. The nature of the lesion has, however, varied in different cases. In some the vessels have been congested or hemorrhages have occurred; in others atrophy of nerve cells or of nerve fibres has been found. Subserous hemorrhages on the surface of the fourth ventricle were observed by Bruhl and by Lasvènes. Numerous hemorrhages were found by Hale White [149] in the medulla in one case. These hemorrhages extended in a vertical direction from the nucleus of the sixth nerve up to the lower part of the Sylvian aqueduct. Laterally they extended from the middle line as far as the restiform bodies, which were also slightly implicated. They were

superficial and nearly all were situated in the posterior part of the reticular formation. Hale White considers that these hemorrhages were evidence of pre-existing disease of the floor of the fourth ventricle, as a result of which the hemorrhages occurred during the attack of pneumonia which immediately preceded death. Müller[147] found recent ecchymoses in the floor of the fourth ventricle in three cases. Hemorrhages have also been described in the same situation by Grainger Stewart and Gibson, by Greenfield, and by R. Martin. Müller does not consider these hemorrhages to be of any pathological importance, and it must be remembered that similar hemorrhages are not infrequently found in cases in which no symptoms of exophthalmic goitre have occurred. In an uncomplicated case of three years' duration in which death occurred from pleurisy, Mendel[150] found atrophy of the left restiform body, a diminution of nerve fibres, and increase of connective tissue with atrophy of the right solitary bundle. In a case of locomotor ataxy with exophthalmic goitre, Marie and Marinesco[15] found, in addition to the usual changes which are present in locomotor ataxy, a degeneration of the whole of the solitary bundle on each side and of the bulbar portion of the ascending root of the fifth nerve. These authors draw attention to the fact that though degeneration of the solitary bundle was found by Oppenheim in a case of locomotor ataxy, the patient exhibited some of the symptoms of exophthalmic goitre during life, while in another case of locomotor ataxy in which such symptoms were absent the solitary bundle was normal. A definite lesion of this nature is important, especially when we consider the relationship of the solitary bundle to the vagus nerve. A few other slight changes have been described in the medulla, but as they have occurred in almost isolated cases they were most probably accidental and of no pathological importance. The real significance of either hemorrhages or degenerative changes in the medulla cannot, however, be fairly estimated until a larger number of cases has been examined, for Müller, who has examined this region carefully in three cases, and other observers have failed to detect any signs of degeneration in it. Möbius also maintains that, as a rule, no lesion of the medulla is found in cases of exophthalmic goitre.

Spinal Cord.—Slight changes have been described in the spinal cord in a few cases, but they do not appear to have any special relationship to the disease under consideration.

Sympathetic System.—Changes in the cervical ganglia have been described by several writers, and at one time exophthalmic goitre was commonly supposed to be a disease of the sympathetic nervous system. The changes most commonly observed are subacute inflamma-

tion of the ganglia with infiltration by leucocytes and degeneration of the nerve cells. It has, however, been shown more recently by Hale White and others that fatty and pigmentary degeneration and atrophy of the nerve cells, as well as infiltration with leucocytes and increase of interstitial connective tissue, may occur in the sympathetic ganglia of the adult both in health and in the course of various diseases, and that consequently such changes are of no pathological significance. Then, again, in some cases of exophthalmic goitre the sympathetic system is found to be quite normal in structure. Hamar,[152] who has examined twenty-two cases of exophthalmic goitre, found the sympathetic was normal in fifteen. It is thus evident that there is no constant or essential lesion of the sympathetic system in exophthalmic goitre.

Heart.

In many cases the size of the heart is unaltered. In some there is dilatation, and occasionally hypertrophy, of the ventricular walls, but only to a moderate degree. Valvular disease is rarely seen.

Orbit.

The amount of fat in the orbit is generally increased, and this has been supposed to contribute somewhat to the exophthalmos, though most probably the dilatation of the vessels of the orbit also helps to push the eyeball forward.

Lymphatic Glands.

Swelling of the lymphatic glands has been found in some cases.

Pathology.

No good purpose would be served by a full consideration of the various theories of the pathology of exophthalmic goitre which have found favor in the past, as they have proved to be untenable. Thus some of the early observers considered that disease of the heart and blood-vessels was the essential morbid change. When, however, it was repeatedly found that these organs were not diseased, the abnormal action of the heart was supposed to be due to a cardiac neurosis, a view which has also been abandoned in the light of more recent research. Only those theories which, in the present state of our knowledge, appear to be capable of explaining the phenomena of the disease will now be considered.

It is an interesting fact that until quite recently the share which the enlarged thyroid gland might have in the production of the other

symptoms was almost entirely left out of consideration. The only symptoms which were attributed to the thyroid gland were those which were supposed to be caused by the mechanical pressure of the enlarged gland upon the surrounding parts.

The most important theories which have been advanced to explain the symptoms of exophthalmic goitre may most conveniently be divided into those which are based upon some observed or supposed lesion of the nervous system, and those which take the abnormal condition of the thyroid gland itself as the primary cause of the other morbid phenomena which occur in the course of the disease. With regard to the first group, it must be added that almost every part of the nervous system has at one time or another been considered to be the seat of the essential lesion in exophthalmic goitre. In the case of the brain, the spinal cord, and the peripheral nerves the evidence is so slender that we need not consider it further. The most important theories have attributed the symptoms to a diseased condition either of the sympathetic system or of the medulla oblongata, and these must be considered separately.

Sympathetic Nervous System.

Koeben [109] advanced the view that the sympathetic nerve was pressed upon by the goitre and that this led to the symptoms of the disease. In answer to this, however, it was pointed out that the enlargement of the thyroid gland may be slight and that the symptoms of exophthalmic goitre are not produced by large goitres. Many observers have considered that the symptoms might be caused by an irritant or paralytic lesion of the sympathetic or by a combination of the two. There are, however, strong objections to all these views.

Thus although exophthalmos can be produced experimentally by stimulation of the sympathetic, it does not reach the same degree (Vulpian [153]) as in many cases of exophthalmic goitre. Then stimulation of the sympathetic causes dilatation of the pupil, which is rarely seen in exophthalmic goitre; and finally, the continuance of an irritant lesion for years, even if possible, is highly improbable. Boddaert [173] by section of the cervical sympathetic and ligature of the jugular vein produced a *temporary* exophthalmos as a result of the altered venous circulation, but this symptom is often of long duration in exophthalmic goitre. We know of no lesion which can produce stimulation and paralysis at the same time for many months. We have already seen that in many cases of exophthalmic goitre the sympathetic system has been found to be quite healthy, and that such

changes as have been observed in others may also occur in healthy persons. Thus there is no anatomical or physiological basis for the view that the sympathetic nervous system is primarily at fault in exophthalmic goitre.

Medulla Oblongata.

Much evidence has been brought forward to show that exophthalmic goitre is essentially due to either functional or organic disease of the medulla oblongata. The part played by the medulla may be either primary or secondary and may take effect in three different ways. According to one theory the disease is due to an organic lesion of the medulla; other observers consider that though the medulla is the primary seat of the disease there is only a functional derangement and no organic disease; a third and, as we shall see, a very reasonable view is that the nervous symptoms are due to a perverted action of the nerve centres in the medulla, which occurs secondarily to the change in the thyroid gland, and as a result of the action of increased or abnormal thyroid secretion upon these nerve centres. Charcot and Trousseau both considered that exophthalmic goitre was a functional neurosis, basing their opinion upon the absence of a definite organic lesion and upon the similarity between some of the characters of the disease and those of other neuroses. Mackenzie [120] has drawn attention to the similarity between the symptoms of fear and those of exophthalmic goitre, and has advanced the ingenious theory that the affection is a prolongation of the condition of fear owing to a failure of the nervous system to recover its normal condition after a shock. Those who maintain that the disease is due to an organic lesion of the medulla base their opinion upon the results of experimental lesions of this part of the nervous system and upon the structural changes which have been described by various observers. Filehne [154] found that section of the anterior fourth of the gray matter of the restiform bodies was followed by exophthalmos. In some experiments both enlargement of the thyroid gland and exophthalmos occurred, and in one tachycardia was also produced. Dardufi [155] after section of both tubera acustica observed tachycardia together with widening of the palpebral fissure and slight exophthalmos. Bienfait [156] made bilateral transverse sections through the gray matter of both restiform bodies in rabbits. This was followed by marked alteration in the cardiac rhythm and a fine regular tremor. Exophthalmos was present in rather more than one-third of the animals, and in one-fourth of them distinct hyperæmia of the thyroid gland developed. Interesting and important as these experiments are, they do not appear to prove that an organic lesion of the medulla

is necessary to produce exophthalmic goitre, any more than the production of experimental glycosuria in a rabbit by puncture of the medulla oblongata proves that diabetes is due to disease of the nervous system. They do show, however, that some of the symptoms of exophthalmic goitre may occur as a result of interference with the normal action of the nerve centres in the medulla. Mannheim [116] mentions an interesting case in which exophthalmic goitre developed a few days after the onset of a bulbar hemorrhage and improved as absorption of the blood clot took place. It must, however, be remembered that in the great majority of cases no lesion has been found which could be at all compared to that made in the experiments or to a bulbar hemorrhage sufficiently large to produce hemiplegia as in Mannheim's case. The strongest argument against the theory of an organic lesion is the result of post-mortem examination, which, as we have already seen in many cases, reveals no morbid change in the medulla, and in those in which changes have been described the latter have varied so much in their nature that they are more likely to be secondary than primary in origin. For the view advanced by Hale White that the structural alteration in the medulla may be too fine to be detected by our methods of examination there is as yet no sufficient basis.

Thyroid Gland.

During the last ten years our knowledge of the thyroid gland has been considerably increased, and much more attention has been paid to its condition in exophthalmic goitre and to the general effects which may occur as a direct result of thyroid disease. While considering the pathology of myxœdema we reviewed the facts which have led to the conclusion that the thyroid gland is an important secretory gland, the secretion from which ultimately enters the blood by way of the lymphatics. This secretion is necessary for the maintenance of health, and the important part which it plays in the normal metabolism of all parts of the body, and especially of the nervous system, is shown by the disastrous results which occur when it is lost, and which are so clearly seen in myxœdema, cretinism, and cachexia thyreopriva. We have seen that the structural changes which have been described by Greenfield [148] and others in the thyroid gland in exophthalmic goitre clearly show that there is a great increase in the amount of actively secreting gland tissue. The microscopical appearances indicate that not only is the secretory activity much greater than usual, but also that the rate of absorption is probably much increased. The condition of the gland may be fairly compared to that of the mammary gland during lactation, and we may reasonably

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conclude that a far larger quantity of thyroid secretion is poured into the circulation than in health. Whether the secretion formed under these circumstances is of the same composition as it is in health or not we do not know. It is quite possible and indeed probable that the secretion is not only increased in quantity but also altered in character. Further investigation, however, into the nature of normal thyroid secretion is required before this point can be determined. When we consider that fibrosis and atrophy with loss of function of the thyroid gland lead to such widespread changes as we see in the different varieties of myxœdema, we may naturally expect that hypertrophy and increase of activity in the same gland will also produce important effects of a different and in the main opposite character. In fact, it seems very probable that the abnormal condition of the thyroid gland is as important a factor in exophthalmic goitre as it is in myxœdema. Möbius [132] was the first to suggest and ably support this theory of the pathology of exophthalmic goitre which with modifications has received support from Wette,[171] Müller,[147] Bramwell,[118] Maude,[128] Greenfield,[148] and Marie. Briefly stated, then, we maintain that in exophthalmic goitre there is an excessive formation and absorption of thyroid secretion which may or may not be abnormal in character, and that the symptoms of the disease are due to the presence of this excess of secretion in the blood and to its action on the tissues, especially upon the nerve centres in the medulla oblongata and elsewhere.

We hope to show that this theory is capable of explaining the great majority of the symptoms of exophthalmic goitre. Some maintain that although many of the symptoms may be due to the thyroid gland, there are others, such as the exophthalmos, which require some further explanation. On this point we can as yet come to no final conclusion. When we contrast and compare the clinical symptoms of myxœdema and exophthalmic goitre we find that in each disease the same organs are liable to be affected, but in opposite ways. Both diseases are more frequent in the female than in the male sex. The same organs—viz., the thyroid gland, the nervous system, the heart, and the skin—are principally affected in each. In fact, knowing that myxœdema is due to diminished activity of the thyroid gland, we find in exophthalmic goitre just the symptoms which we might expect to be produced by excessive activity of the gland, for the one disease presents a remarkable contrast to the other. In myxœdema there is fibrosis with atrophy of the thyroid gland; in exophthalmic goitre increase in size and hypertrophy of the gland. In the former there is stolidity, lack of interest, and bodily inactivity; in the latter, excitability, over-anxiety, and restlessness. In myxœdema

the action of the heart is weak and slow; in exophthalmic goitre the contractions of the heart are strong and very frequent. The skin in myxœdema is pale, thickened, and rough, it feels cold and dry, perspiration is absent, and the electrical resistance is increased; whereas in exophthalmic goitre the skin is often flushed, thin, and smooth, it feels warm and moist, perspiration is excessive, and the electrical resistance is diminished. In myxœdema the temperature is subnormal; in exophthalmic goitre it is frequently above the normal level.

Other significant facts which lend support to the thyroid origin of the disease are the beneficial results which have followed medical and surgical methods of treatment which tend to diminish the size or to lessen the excessive functional activity of the thyroid gland. Belladonna is of great service in the treatment of the disease, probably because it diminishes thyroid secretion. Belladonna checks secretion in other secretory glands, and there appears to be no reason why it should not do the same in the thyroid gland, the activity of which, as has been shown by Wyss, can be considerably increased by pilocarpine. The inunction of the red iodide of mercury ointment over the enlarged gland reduces the size of it in many cases and causes marked diminution of the symptoms. Operative treatment, such as ligature of some of the thyroid arteries or excision of part of the enlarged gland, has in some cases been followed by cure of the disease, and in others by great improvement in the symptoms. The results of this method of treatment will be considered more fully in dealing with the treatment of the disease.

Valuable evidence is given by the effects which are produced in man by excessive doses of thyroid extract, for it has been repeatedly observed that they are identical with some of the chief symptoms of exophthalmic goitre. Patients who have had myxœdema appear to be specially susceptible to the action of large doses of thyroid extract, so that a condition of thyroidism may be produced, either, as the writer [160] has found, by prolonged administration of doses only slightly in excess of what is suitable for the patient or, more rapidly, by giving unusually large doses. The most frequent symptom produced by large doses of thyroid extract is increased force and frequency of the heart's action. The patient complains of palpitation, and the heart beats strongly at the rate of 100 to 120 or 130 beats a minute. A fine tremor, just like that of exophthalmic goitre, may also occur. The skin is flushed and moist, and if the administration of the extract is prolonged emaciation may follow. In addition to these the usual results of the administration of excess of thyroid extract, elevation of temperature, sleeplessness, restlessness, polyuria, albuminuria, incomplete paraplegia, sensations of heat, and

diarrhœa, all of which occur in exophthalmic goitre, have been observed by Marie.[157] Béclère[158] has seen exophthalmos, increased rate of respiration, and transient tremor of the arms develop, in addition to the other symptoms already mentioned, in a woman who had suffered from myxœdema and who by mistake had taken ninety-two grams of thyroid gland in eleven days. Thus with the exception of v. Graefe's symptom and Stellwag's sign all the common and many of the less frequent symptoms of exophthalmic goitre have occurred as a result of the administration of excessive doses of thyroid secretion. Some of these symptoms have been produced in healthy individuals, for G. F. Johnston[159] found that after taking from four to nine thyroid tablets each day there was acceleration of the pulse from 70 to 120, accompanied by palpitation, flushing, tremors, sensations of heat, and perspiration.

The effects produced by the treatment of exophthalmic goitre by thyroid extract have varied in different cases and no very definite conclusions can be drawn from them. One interesting case has been recorded by A. G. Auld[161] in which as a result of this treatment the exophthalmos was increased, and the pulse was accelerated from 99 to 138. A rise of temperature took place and was followed by diarrhœa, sickness, perspiration, and emaciation. Similar results have also been recorded by Kocher.[162] Another important fact which has been repeatedly observed is that recovery from exophthalmic goitre may subsequently be followed by the onset of myxœdema. Such cases have been recorded by Gowers,[123] Corkhill,[174] Ord,[163] Bowles,[163] and Gowan.[175] Ord has seen myxœdema develop when the enlarged thyroid gland has been too rapidly reduced in size by treatment. The explanation of this sequence appears to be that a chronic interstitial thyroiditis is set up, which first of all cures the exophthalmic goitre by diminishing the size and functional activity of the thyroid gland, and then by further progress leads to an atrophy and loss of function of the gland, which is followed by myxœdema. To put it briefly the evidence afforded by (1) the microscopical appearances of the thyroid gland, (2) the contrast between exophthalmic goitre and myxœdema, (3) the beneficial effects of medical and surgical treatment of the enlarged thyroid gland, (4) the various effects which are produced by the administration of thyroid extract in health, in myxœdema, and in exophthalmic goitre, and (5) the occurrence of myxœdema after recovery from exophthalmic goitre, indicates that many if not all the symptoms of the disease may be due to excessive or perverted activity of the thyroid gland.

In opposition to this view it has been urged that myxœdema and exophthalmic goitre may occur simultaneously in the same individ-

ual. This I have never seen, nor do I know of any case in which this combination has been observed in this country. Of the two cases which have been published by Sollier [164] one cannot be regarded as a case of myxœdema, as the patient was to my knowledge subsequently treated by thyroid extract with little or no diminution of the swelling which had been considered as myxœdematous. Another objection is that the symptoms of exophthalmic goitre may occur without appreciable enlargement of the thyroid gland. In answer to this it may be mentioned that such cases are not common, and that the more closely cases are observed the more frequently is enlargement of the gland detected at one time or another. Considerable enlargement has been found after death when little or none could be observed during life, so that not only may there be enlargement which is not detected during life but it is quite possible for hypersecretion and rapid absorption to take place without marked enlargement. In conclusion, it must be mentioned that, as Greenfield [148] has pointed out, the changes which have been described in the medulla oblongata resemble those which are found in rabies and tetanus, and so, like them, may be entirely secondary in origin.

As yet we know as little of the exciting cause of the change in the thyroid gland in exophthalmic goitre as we do in myxœdema.

Diagnosis.

In well-marked cases of exophthalmic goitre in which the three chief symptoms, enlargement of the thyroid gland, rapid pulse, and exophthalmos are all present, the diagnosis presents no difficulty, as the appearance of the patient at once suggests the nature of the disease. At the commencement of the illness, especially when the onset of the disease is gradual, there may for a time be difficulty in arriving at a correct conclusion as to the nature of the case. As the symptoms become more developed their true nature soon becomes evident. In ill-defined or "fruste" cases, as they have been termed, there may be a rapid pulse but no exophthalmos, and no enlargement of the thyroid gland which can be detected by palpation at the time when the patient is seen. In some of these cases it is difficult to arrive at a correct diagnosis, especially after a single examination. If the patient is under observation for a time, other symptoms, such as slight temporary enlargement of the thyroid gland or tremor, may be observed at one time or another and indicate that the rapid action of the heart is due to exophthalmic goitre. It is generally agreed that no case without acceleration of the pulse can be considered as exophthalmic goitre. There is generally little difficulty in distin-

guishing the enlargement of the thyroid gland in exophthalmic goitre from an ordinary goitre. In goitre the thyroid gland is usually larger and firmer in consistence than in exophthalmic goitre. Pulsation and thrill are generally absent. We have seen that exophthalmic goitre is usually accompanied by various general symptoms. In ordinary goitre these are absent. It is important to remember, however, that serious symptoms may be produced by the mechanical pressure of a large goitre upon the surrounding parts. Such symptoms are, however, readily distinguishable from those which occur in exophthalmic goitre. Broadly speaking, the symptoms of exophthalmic goitre are toxic in origin, while those of ordinary goitre are due to local mechanical pressure. In some cases the symptoms of exophthalmic goitre may appear in a patient who has for long had an ordinary goitre. In such a case the history of simple enlargement of the thyroid gland, without symptoms, preceding the onset of the rapid pulse and other signs of exophthalmic goitre, will enable a correct diagnosis to be made. The rapid pulse of exophthalmic goitre differs from that of functional palpitation by its persistence. In palpitation the pulse rate sinks to normal between the attacks, but in exophthalmic goitre the heart at all times beats more rapidly than in health. The rapid pulse of organic heart disease is distinguished by the presence of the physical signs of the disease. When organic heart disease occurs in exophthalmic goitre the presence of the latter must be determined by the other symptoms which are present.

Prognosis.

The prognosis in an early case of exophthalmic goitre is by no means easy. In young patients the prognosis is more favorable than in those who have reached middle age, and it is more favorable in women than in men. Social position must also be taken into account, as those who are able to afford rest and quiet with proper attention are more likely to do well than those in poor circumstances. Beyond these factors the prognosis has to be founded upon the severity of the symptoms, the mode of onset, and the duration. Rapid onset of severe symptoms is of serious import, though, as we have seen, rapid onset may be followed by rapid recovery. The appearance of signs of cardiac failure, such as dyspnœa and œdema, indicates that there is danger. Vomiting, diarrhœa, rapid emaciation, and loss of strength warrant a grave prognosis. In an ordinary case with no very severe symptoms a fairly good prognosis may be given, though it is very difficult to tell how long the disease will continue and whether complete or only partial recovery may be expected. If

treatment is soon followed by improvement the prognosis is better than when the symptoms remain stationary.

Treatment.

Many different lines of treatment have been adopted for exophthalmic goitre according to the current views of the time as to the nature of the disease. Thus in addition to variations in general treatment special attention has been directed at one time to the heart, at another to the nervous system, and lately to the thyroid gland. It will be most convenient to consider separately general hygienic treatment, medicinal treatment, treatment by electricity, and surgical treatment.

General Hygienic Treatment.

We have seen how susceptible patients are to the effects of mental excitement and bodily exertion under the influence of which the general nervousness is increased and the pulse is accelerated. It is therefore important that all sources of such excitement and exertion should be removed, and that the patient should lead as quiet and as easy a life as possible. If such conditions cannot be fulfilled at home, a visit to the country for several weeks or months is most beneficial. High altitudes as a rule are not suitable, but many cases do very well at the seaside if the air is not too bracing. No special health resort need be selected, nor is it advisable to send a patient to a regular watering-place, where large numbers of people are gathered together, for it is only in isolated cases that good results have been obtained by taking a course of medicinal water. If the patient is a good sailor, a sea voyage undertaken under favorable conditions may do much good. A considerable portion of the day may be spent out of doors, but only gentle exercise should be taken and several hours of each day may with advantage be spent lying down. In severe cases complete rest in bed for a time is necessary, and this alone may reduce the frequency of the pulse by thirty or forty beats a minute. The diet must be light and digestible, and milk should form an important part of the daily food. Alcoholic stimulants are best avoided unless specially indicated by cardiac weakness. Tea, coffee, and tobacco must be used in great moderation if at all. Baths have been found to be of service by some, and in cases where nervousness is excessive warm baths or shower baths at 100° F. may be employed, the effect being carefully watched. Massage in moderation is useful in cases where absolute rest in bed is necessary, but it is not required if gentle exercise can be taken. Gymnastic exercises have been recommended

but do not appear to be suitable, though the passive mechanical exercises as employed by Bryson have yielded good results.

Medicinal Treatment.

Medicinal treatment may have as its object either the cure or improvement of the disease as a whole or the relief of special symptoms. In the first place treatment must be directed toward improving the condition of the thyroid gland. We must, if possible, reduce the size of the gland, diminish its functional over-activity, and counteract the toxic effect of the excessive or abnormal secretion upon the nerve centres. The most efficient means of reducing the size of the gland is inunction by the red iodide of mercury ointment. This treatment has for long been employed by my father, Dr. William Murray, with very good results. A small piece of this ointment about the size of a pea should be well rubbed into the skin over the enlarged thyroid gland each night till the skin becomes too tender for the patient to bear the further application of the ointment. After an interval of a few days or a week the treatment may be resumed and continued in this manner at intervals for several weeks or months at a stretch. Exposure of the skin to the direct rays of the sun has been found to increase the efficiency of this treatment in the case of parenchymatous goitre, so that when practicable it is better to apply the ointment in the morning and expose the skin to the sun's rays. In any case the skin of the front of the neck should be left exposed to the light as much as possible, as advised by Turner.[167] In this way a marked reduction in the size of the gland is brought about in many cases, with a corresponding improvement in the other symptoms. As an alternative, the skin over the gland may be painted with tincture of iodine, or an ointment consisting of half a drachm of extract of belladonna and one ounce of iodine ointment may be applied on lint to the skin over the gland each night. Of the many drugs which have been employed in the treatment of this disease belladonna has proved to be the most useful, presumably by diminishing the over-activity of the thyroid gland. Either the tincture or the extract of belladonna may be used, though the former is generally preferred. Ten minims of the tincture of belladonna may be given three times a day, and the dose gradually increased until the physiological effects are produced and dilatation of the pupil and dryness of the mouth and throat indicate that sufficiently large doses are being given to appreciably diminish secretion. As much as fifteen or twenty minims given three times a day may be necessary to produce the desired effect. Atropine may be given instead in doses of $\frac{1}{250}$ grain in a pill. Potassium bromide is

also of great service, especially in lessening the general nervousness and the excitability of the nerve centres. As it acts in a different manner to belladonna, it may with advantage be combined with it in ten or fifteen grain doses three times a day. A larger dose at night is useful if there is insomnia. Sodium phosphate, in daily doses of from half a drachm to two drachms and a half in water, has been recently recommended by Trachewsky, and Kocher [162] speaks highly of the good results he has obtained by its use. Arsenic and strychnine are both very useful in some cases and especially when given in combination. Ergot has often proved to be beneficial. Digitalis and strophanthus have little effect in slowing the pulse, but are of service if the heart shows signs of failure. Iron and quinine improve the condition of the patient by acting as general tonics, the former being specially indicated if there is anæmia. The ammoniated tincture of valerian was found to be of service in some cases by the late Sir George Paget when other drugs had failed. Tincture of iodine or the iodides have proved beneficial in some cases, but harmful in others, as the palpitation, headache, and feeling of heat have been increased by their use. Thyroid extract has occasionally been used to advantage. As, however, it has seriously aggravated the symptoms in some cases, as might be expected, it must be used with caution. The thymus gland has been used in one case of twenty years' duration in a man by Owen,[165] and in another in a woman by Mikulicz,[166] with remarkably good results. In the former case one lobe of the cervical portion of the thymus taken three or four times a week was found to be a useful dose, in the latter ten grams of finely mixed sheep's thymus were given three times a week and the dose gradually increased up to twenty-five grams. Other drugs which have been employed in the treatment of exophthalmic goitre are sodium salicylate, aconite, and veratrum viride. Any nasal, uterine, or other local disease which may act as a source of irritation must be treated by appropriate remedies. Inflammation of the eyeball requires prompt attention. If the palpitation is severe and distressing to the patient it may be controlled by the application of an ice-bag to the precordial region. Hip baths and the application of mustard to the feet are useful, or a belladonna plaster may be worn over the cardiac region. Marked anæmia may be treated by arsenic or iron, but they often fail to give relief. Failure of the heart must be met by the exhibition of digitalis, strophanthus, or strychnine, and diffusible stimulants. Severe dyspnœa has been relieved by venesection or the application of leeches, but if due to pressure of the enlarged thyroid gland an operation may be necessary to relieve it.

Electrical Treatment.

Galvanism has long been used in the treatment of exophthalmic goitre. The best method of application is that recommended by Cardew.[168] The anode with a diameter of three inches is well moistened with a warm solution of salt in water and placed on the back of the neck over the seventh cervical vertebra. The cathode, which should measure one inch and a half in diameter, is moved up and down along the line of the anterior border of the sterno-mastoid muscle, first on one side of the neck and then on the other. A current of two or three milliampères should be employed for six minutes at a time thrice daily.

The faradic current has been used successfully by Vigouroux,[169] who recommended that the interrupted current should be employed in the following manner: The positive electrode, which should have a diameter of about four inches, is to be well moistened and applied to the nape of the neck all the time the current is passing. The negative electrode, shaped like an olive with a diameter of half an inch, must be pressed firmly into the neck over the carotid artery at the anterior border of the sterno-mastoid muscle, on a level with the angle of the jaw, for one minute and a half on each side. The current should be strong enough and cause contraction of the sterno-mastoid muscle. The orbicularis palpebrarum is then faradized on each side. The small electrode is then replaced by one two inches in diameter which is applied over the enlarged thyroid gland. This same electrode is next placed over the heart at the inner end of the third left intercostal space and the current is reversed. The whole sitting lasts for ten or twelve minutes and should be repeated every other day for from six to twelve months. Few have obtained such good results as Vigouroux himself, and some have failed to see any improvement follow the electrical treatment. The good results which sometimes follow its use show that it is well worth trying, though the difficulty of carrying out the treatment thoroughly for such long periods is likely to prevent its adoption in many cases.

Surgical Treatment.

Surgical treatment may be undertaken in exophthalmic goitre for several different reasons. An operation is necessary when the enlarged thyroid gland compresses the trachea sufficiently to produce dyspnœa and stridor, which other measures have failed to relieve. If these symptoms are urgent, part of the enlarged gland should be removed. In one such case Sir Joseph Lister[170] removed both lobes

and probably left the isthmus with good results. Division of the isthmus has been recommended in such cases, but practically it is not often feasible, and removal of one lobe is better.

If part of the enlarged thyroid gland is cystic the cyst should be tapped, or tapped and injected with solution of perchloride of iron. If the cyst fills again it should be incised and drained. If this does not succeed, it should be enucleated. The importance of thus treating any cyst which is present in the gland is shown by the improvement in the general symptoms which follows in many instances. I have seen the symptoms of exophthalmic goitre almost entirely disappear after incision and drainage of a cyst in the enlarged gland.

Möbius [132] mentions one case published by Dubmeil in which the symptoms gradually disappeared after enucleation of the cyst, and another in which removal of a cystic adenoma by Rupprecht was followed by equally good results.

Surgical treatment may be undertaken to reduce the size of the goitre and so to relieve the other symptoms which are due to the hypertrophy and hypersecretion of the gland. Treatment with this object in view may be carried out in various ways. The injection of iodine has been employed as for parenchymatous goitre, but it is not free from danger. Three operations are employed to reduce the size of the gland: 1. Ligature of some of the thyroid arteries so as to diminish the blood supply and cause atrophy; 2. Removal of a part of the enlarged gland; 3. Exposure of the enlarged gland to the air so as to induce atrophic changes in its substance. Surgical treatment has been carried out with success in a considerable number of cases. As such treatment is not free from danger to the life of the patient, and is still on trial, very careful judgment must be exercised in determining whether any given case is suitable for operation or not. We cannot as yet estimate the true value of the operative treatment of exophthalmic goitre, but we do not consider that its utility is as yet sufficiently proved for it to be adopted as a routine method of treatment. We shall, however, endeavor to indicate the circumstances under which an operation may be advised. No operation having for its object the reduction in size and activity of the thyroid gland should be undertaken until the local and general means of medical treatment at our disposal have been fully tried and have failed to relieve the patient sufficiently. When the symptoms are not severe and do not tend to become worse, we also think that it is best to continue medical treatment and not to advise any operation. In very advanced cases in which there is cardiac degeneration and much emaciation there is a very considerable risk of the operation itself proving fatal. In such cases the actual cause of death is by no means

evident, but fatal syncope has occurred both during the operation and shortly after it, so that death may be due to the influence of shock upon an enfeebled nervous system or to the action of the anæsthetic upon a degenerated heart. In one of my cases which is represented in Fig. 49, the disease had been fully developed for about a year. There was considerable enlargement of the thyroid gland, exophthalmos, a pulse varying from 124 to 140, hypertrophy of the heart, tremor, sweating, emaciation, and diarrhœa. Local and general medical treatment had been of no avail, so I advised an operation. F. Page removed the right lobe of the thyroid gland, which was rather larger than the left, the patient being anæsthetized by ether. The operation was rapidly performed and no excess of blood was lost. The patient remained cyanosed after the operation and died suddenly an hour later. No autopsy was allowed, so that the immediate cause of death was not explained. This is the only one of my cases in which part of the gland has been removed. The most suitable cases for operation appear to be those in which the symptoms are of moderate or great severity but not of very long duration, and in which the symptoms became worse in spite of all medical treatment. Buschan [142] considers that cases in which the enlargement of the thyroid gland has existed for some time previous to the development of the other symptoms are specially suitable for operative treatment. It is also important that the heart and nervous system should show no signs of degeneration. Under these circumstances operative treatment may be advised as offering the best means of effecting a cure or of greatly diminishing the severity of the symptoms and of improving the general condition of the patient. Such a conclusion seems to be justified by the good results which have been obtained in a majority of the cases treated by operation. From the records which are at present available, it is difficult to decide definitely which operation is the most suitable.

Partial thyroidectomy, as recommended by Wette,[171] has been carried out in the majority of cases, and is beneficial not only on account of the immediate removal of some of the superabundant thyroid tissue, but also because of the partial atrophy which is nearly always induced in the remaining portion of the thyroid gland. It appears also that improvement takes place more rapidly after partial thyroidectomy than after ligature of the thyroid arteries. Kocher,[162] who is a strong advocate of the operative treatment of exophthalmic goitre, ligates the three largest thyroid arteries, unless some special condition renders this difficult, in which case he removes one lobe of the gland. He considers that ligature of the thyroid arteries is a much more efficient method of bringing about an atrophy of the

thyroid gland than partial thyroidectomy, and that it is the right operation to perform in most cases. It is not possible as yet to decide between the respective merits of these two operations, though ligature of the arteries seems to be the less serious proceeding and the more suitable if the enlarged gland is very vascular.

Exothyreopexy as practised by Poncet consists in exposing and fixing the enlarged thyroid gland in the wound and leaving it to shrink under an aseptic dressing. This method of operating is apparently no less dangerous than the others, and it does not seem to be so efficient. It has, however, been carried out in only a few cases. In a successful case the improvement which follows the operation is gradual, and in some cases there is an aggravation of the symptoms at first, for the operation has been followed by great prostration, rapid pulse, dyspnœa, vomiting, insomnia, pneumonia, pleurisy, and laryngeal paresis. These serious symptoms generally pass off after a few days when real improvement may begin. The improvement is the more likely to be slow if the symptoms have lasted for a long time. In a few the improvement has been rapid and the exophthalmos has sometimes disappeared almost directly after the operation. In most cases the earliest sign of improvement is a diminution of the palpitation and a gradual lessening of the frequency of the pulse. The patient then becomes less excitable and is able to sleep better. The strength is gradually regained. The tremor, feeling of heat, and perspiration may disappear a few weeks or months after the operation. The exophthalmos is often one of the most persistent symptoms, but in some cases all the symptoms disappear in time, though months or even years may elapse before the full benefit of the operation is felt. The amount of success which has followed the operative treatment of exophthalmic goitre is best illustrated by the tabulated cases of various authors. Kocher [162] has operated on 34 out of 39 cases which have been under his care. Of these 3 died, 1 from the operation itself and 2 from embolism. In all the others improvement or cure took place after a longer or shorter period. In 900 partial thyroidectomies for goitre Kocher has had a mortality of twelve per cent., so that it would appear that the operation is less dangerous for exophthalmic goitre than for ordinary goitre. Mikulicz has also had good results, and finds that patients who have been operated upon are greatly pleased with the progress they afterwards make. Stierlin has collected the reports of 29 cases in which an operation had been performed, and in 22 recovery had taken place. Putnam [172] has tabulated 51 cases, in 4 of which death occurred as a result of the operation. In nearly all the others improvement, and in many recovery, took place. Buschan [142] has collected 80 cases of exophthal-

mic goitre which have been treated by operation; of these 31 were said to be cured, 20 improved, 16 derived no benefit from the operation, 6 died from the operation; in the other 7 the result was not known. When we consider that probably nearly all these cases had failed to derive benefit from medical treatment before the operation was performed, the results are most encouraging, and indicate that an operation offers a good prospect of affording relief under the circumstances which we have already briefly outlined.

GOITRE.

(Guttur, the throat.)

Synonyms.—Bronchocele, thyreocele, thick-neck, Derbyshire-neck. French, *Goître;* German, *Kropf, Struma.*

Definition.

Under the name of goitre we shall include all enlargements of the thyroid gland which are not due to inflammation, malignant disease, exophthalmic goitre, or the presence of animal or vegetable parasites.

Etiology.

Goitre may occur either sporadically or more usually in an endemic form. Epidemics of goitre have occasionally been observed, as at Serdobol in Finland, in which Sievers [176] records that a teacher and fifteen children were suddenly attacked. Of the exciting cause of sporadic goitre we have no definite knowledge. Endemic goitre occurs under certain local conditions to which it undoubtedly owes its origin, as removal from the goitrous district prevents the development of the disease. These special conditions we shall consider separately.

Sex.—Women are more liable to suffer from goitre than men. The exact proportion between the two sexes is very difficult to determine, as the proportion in different parts of England has been found to vary from seven to one up to forty-four to one. The difference in the liability of the two sexes in this country is said to be due to the fact that women drink more water than men. In India, where men and women both drink water, the two sexes are equally affected.

Heredity.—In some families goitre is certainly hereditary, in others an apparent heredity is really due to different members of the family being subject to the same local conditions.

Age.—Congenital goitre occurs sometimes, but it is rare. The disease does not often develop during childhood, but generally after the age of puberty; it may develop in persons over fifty years of age.

Locality.—Goitre occurs in almost all countries, but the endemic form is confined to certain districts, many of which resemble each other in their natural configuration. For the most part endemic goitre is found in mountainous districts, in the deep valleys at the foot of the mountains, but not at higher elevations. In England endemic goitre is found in the Pennine Range in the north (Robinson [177]) and also among the Cotswold Hills (Berry [178]). In Europe we find that in some parts of Switzerland endemic goitre is very prevalent, as in the Valois, and in some parts of the Cantons of Friburg and Berne, as many as eighty or ninety per cent. of the recruits have been found by Bircher [179] to be goitrous. In Italy many cases are found on the southern side of the Alps, as at Aosta. The affection occurs also in the Pyrenees, in Savoy, in the Black Forest, and in Styria. In America Osler [180] shows that although goitre formerly was endemic in certain parts of Virginia, Alabama, and Vermont, it is now rare. In the province of Ontario the disease is prevalent. In Asia goitre is endemic in the Himalayas, in the hilly districts of China, and in the Altai Mountains in Siberia. Goitre may, however, also be endemic in more open country in the neighborhood of rivers with marshy banks, as in the Rhone valley, in Silesia, in North Italy, in the Indian Punjaub, and in the Orinoco valley.

Water-Supply.—Drinking-water, or rather some special constituent of the water, in goitrous districts plays an important part in the causation of goitre. In fact goitre can be produced by drinking such water alone, though we have no proof that all forms of endemic goitre are dependent upon this cause. The importance of drinking-water is shown by the following facts. When a heathy family emigrates into a goitrous district goitre soon develops in some members of the family, whereas removal from the district prevents the further development of goitre in a family in which it has already appeared. When a regiment of young soldiers has moved into a goitrous district, a large number of the men have developed goitre within a few months of their arrival. A change in the water supply of a village where goitre has been prevalent has led to the disappearance of the disease. In other cases an outbreak of goitre has followed the introduction of a new supply of water in a place which was previously free from the disease. The water from certain wells on the Continent is well known to produce goitre, and is successfully used for this purpose by men who wish to acquire a goitre to enable them to be exempted from service in the army. It is thus evident that some kinds of water are capable of producing goitre, though we do not know to what constituent this property is due. It has been supposed that iron pyrites, copper pyrites, sulphate or carbonate of lime,

or carbonate of magnesia in the water may produce this effect, but we have no evidence to show that any one of those substances can produce goitre, nor have we any proof that any special living organism or organic substance can do so either. Whatever the substance may be, it is apparently rendered harmless by heat, as it has been found that such water does not cause goitre if boiled before it is drunk. Other causes which are believed to predispose to the development of goitre are carrying heavy weights on the head, excessive walking up and down hill, and child-bearing.

Symptoms.

The Goitre.—An important characteristic of all goitres, and in fact of all enlargements of the thyroid gland, is that they move upward when the patient swallows. If a goitre is situated low down behind the sternum, it sometimes can be felt only during deglutition. This simple test enables us to distinguish enlargements of the thyroid gland from other swellings in the neck. In parenchymatous goitre there is a smooth, firm enlargement of the whole or of a part of the gland. Adenomata form smooth, well-defined, firm masses in the substance of the thyroid gland. In a fibroid goitre there are hard nodules which project from the enlarged gland causing an irregular outline, so that the original shape of the gland is no longer preserved. Fibroid goitres may reach an enormous size. Cystic goitres are usually globular in shape. A cyst forms a smooth, well-defined, rounded swelling. If the wall is thin, fluctuation can be felt when the cyst is full. If the wall of the cyst is thick, the presence of fluid may be difficult to detect without the aid of an exploratory puncture.

Secondary Symptoms.—When a goitre is small there may be no symptoms beyond the swelling in the neck produced by the tumor. In some cases of goitre one or more of the symptoms of exophthalmic goitre develop. When a goitre is large it may produce serious symptoms by exerting pressure upon the neighboring structures. Pressure upon the veins in the neck may cause over-distention of the veins of the head, with cyanosis and swelling of the face, headache, and drowsiness. The enlarged gland may even compress the arteries, causing convulsions and other symptoms of cerebral anæmia. Pressure upon the nerves of the neck chiefly affects the motor fibres of the vagus which supply the laryngeal muscles. There may be spasm of the glottis. Gradually increasing pressure causes first of all paralysis of the abductor muscles and later complete paralysis of one or both vocal cords. Narrowing of the trachea is often produced by the

pressure of a large goitre and leads to more or less dyspnœa, which at first comes on only after exertion but afterwards may become constant. The goitre may compress the œsophagus and cause dysphagia. Persistent narrowing of the trachea leads in turn to pulmonary emphysema and dilatation of the right side of the heart.

Pathological Anatomy.

The enlargements of the thyroid gland which are classed under the general name of goitre vary considerably in structure. Practically we find they may be divided into general enlargement of the whole gland and special overgrowths of either the glandular, fibrous, or follicular parts of the gland. We thus have (1) Hypertrophic or parenchymatous goitre; (2) Adenoma of the thyroid gland; (3) Fibrous goitre, and (4) Cystic goitre. There are also various combinations and intermediate types. The most detailed account of the varieties of goitre has been given by Wölfler.[181] Hypertrophy of the thyroid gland may cause enlargement of one lobe only or of the whole gland. It may be of congenital origin. Adenoma of the thyroid gland occurs as an encapsuled growth. The size varies considerably. There may be one or more nodules in one or both lobes. The structure resembles that of the gland itself. In a fibrous goitre we have, in addition to overgrowth of the glandular substance, a large increase in the fibrous tissue, which may occur as bands running through the substance of the goitre or as firm nodules. Cystic goitre occurs either as the result of expansion and coalescence of the follicles of an already enlarged gland or as the result of softening of portions of the goitrous tissue. Such goitres often contain a large amount of fibrous tissue and are then distinguished as fibrocystic goitres.

Treatment.

If a patient is living in a goitrous district he should leave it as soon as any enlargement of the thyroid gland develops. A change to the seaside is most beneficial, and if the goitre has been in existence only a short time no further treatment may be necessary to effect a cure. If he cannot leave the district he must drink water only after it has been boiled. The medical treatment of goitre may be either local or general. Local treatment consists of either the application of remedies to the skin over the goitre or the injection of fluids into the goitre itself. Of internal remedies iodine has most frequently been found to be of service. It may be given as potassium iodide in doses of five to twenty grains, or as tincture of iodine in doses of

five to ten minims twice or thrice daily. Woakes [182] recommends the use of hydrofluoric acid. Ten minims of a half-per-cent. solution of the redistilled fluoric acid in an ounce of water should be given twice daily. The dose should be gradually increased. Thyroid extract has recently been shown to be capable of producing a marked reduction in the size of many goitres. Bruns [183] has treated 12 cases of parenchymatous goitre in young people with raw thyroid glands in doses of five to ten grams, twice or thrice a week at first, and once a week afterwards. Of these cases 4 were completely cured; in 5 there was diminution in the size of the goitre, and in 3 there was no result. Kocher [184] has seen considerable reduction in the size of goitres take place under this treatment, but in no case was the goitre entirely removed. He considers that thyroid extract is not superior to iodine, but that it is a useful alternative in the treatment of goitre. Of local applications the most useful is the red iodide of mercury ointment. This should be rubbed into the skin over the goitre daily until the skin is tender, when the treatment should be intermitted for a few days. When possible the skin should be exposed to the rays of the sun after the application. In India this method of treatment has proved to be most successful, and is said to have rarely failed in producing a cure in sixty thousand natives upon whom it was tried by Cunningham.[185] Turner [186] considers that the efficiency of this treatment may be due to the increased facility which iodine preparations give for the passage of the heat rays of the sun to the gland. Thus in any case the skin should be left exposed to the light. Semon [187] has obtained good results by daily applications of a mixture of one part of the unguentum iodi (British Pharmacopœia) with two to three parts of the unguentum potassii iodidi (British Pharmacopœia). In cases of soft goitre the application of liquor epispasticus has been recommended by Morell Mackenzie.[187] These local applications may be used in conjunction with the internal remedies we have already considered. Should they fail, iodine may be injected into the substance of a parenchymatous or fibrous goitre. A solution of one part of iodine in twelve parts of absolute alcohol should be used, and twenty to thirty minims of this may be injected twice a week into the substance of the goitre, a fresh point being selected each time for the injection; great care must be exercised to prevent the wounding of any nerve or vessel in the neck. Very good results are often obtained by this treatment, but there are certain risks, and the injection of iodine has sometimes been followed by sudden death. When a cyst is present in the gland none of the methods which we have considered will effect a cure. Simple aspiration of the cyst is not sufficient, as the cavity soon fills again. A much better method is that which

was practised by Morell Mackenzie.[187] He first tapped the cyst and then injected one or two drachms of a twenty-five-per-cent. solution of perchloride of iron into it. A cyst may also be incised and drained, or, if the cyst wall is thick, it may be excised. Adenomata can be safely enucleated. If all these methods fail to reduce the size of the goitre, and dangerous symptoms occur as the result of pressure, relief may be obtained by division of the isthmus, and if this is not practicable partial thyroidectomy must be performed. For the details of this and the other surgical procedures mentioned, we must refer the reader to works on surgery.

MALIGNANT DISEASE.

Malignant disease is more liable to develop in a previously goitrous thyroid gland than in the normal gland. It may even develop in a goitre which has existed for more than twenty years.

Sarcoma of the thyroid gland is rare, but both round-celled and spindle-celled sarcomata occur in this gland. These growths are very vascular and may bleed very freely if ulceration takes place. Death may occur from direct extension or from secondary tumors in other organs.

Carcinoma of the thyroid gland is also a rare disease except in goitrous districts. It may take the form of epithelioma, glandular carcinoma, or scirrhus carcinoma. The lymphatic glands in the neck are involved early. There is a rare variety in which metastatic pulsatile tumors appear in the bones in various situations. The minute structure of these tumors is identical with that of the thyroid gland itself.

Treatment.

Complete removal of the diseased gland is the only treatment which offers any prospect of relief. It should be performed as early as possible, but even then it is a formidable undertaking.

ACUTE THYROIDITIS.

Etiology.

Acute inflammation of a previously healthy thyroid gland is uncommon, but it occurs more frequently in a gland which is already goitrous. Acute thyroiditis may occur as a primary disease of the thyroid gland, the cause of which is not known, or it may be the result of direct injury to the gland. More frequently it occurs either

as a part of some general infectious process or as a sequela of some acute general disease. Acute thyroiditis occurs in association with, or as a result of, septicæmia, pyæmia, typhoid fever, puerperal fever, diphtheria, rheumatism, measles, and influenza. It has rarely followed pneumonia and bronchitis. Semon[187] mentions one case in which it alternated with orchitis.

Symptoms.

The symptoms of acute thyroiditis are more severe, and lead to greater pressure on the surrounding parts, when the thyroid gland is already goitrous than when it has previously been healthy; otherwise the symptoms are much the same in character in each case. The onset of the inflammation may be rapid, being accompanied by chills or a rigor with rapid rise of temperature. The temperature may rise as high as 104° F. The fever is accompanied by the usual general symptoms which occur in febrile conditions. There is pain in the front of the neck, which may also extend to the face above or to the upper part of the thorax below. There is a sense of constriction of the throat, which in severe cases may be accompanied by difficulty in breathing and in swallowing. In some cases the whole thyroid gland swells, in others only one lobe is affected. The enlarged gland forms a firm tumor in the neck. The skin overlying the enlarged gland may be reddened and tender, and the superficial veins may be engorged. If the swelling of the gland is considerable there is pressure on the surrounding structures in the neck. This is more liable to occur if the thyroid gland has been previously enlarged. Pressure on the blood-vessels may cause headache, and cyanosis of the face. Pressure may also be exerted on the trachea and œsophagus, causing dyspnœa, stridor, and dysphagia. In a favorable case the swelling subsides in the course of a few days or weeks, though some slight enlargement of the gland may persist for some time afterwards. Suppuration may occur. In this case the fever continues, the redness of the skin increases, and fluctuation may be detected beneath it. One or more abscesses may form, which as a rule burst externally. Rarely patches of gangrene may occur, or an abscess may burst into the trachea or mediastinum and cause death.

Treatment.

In the early stages cold should be applied to the front of the neck, either in the form of an ice-bag or ice-poultice or by Leiter's tubes. Leeches may be applied to the skin near the inflamed gland. If the inflammation is not checked by this treatment the skin over the

enlarged gland may be painted with glycerin and belladonna, hot fomentations or poultices being then applied. At the commencement of the attack the bowels should be cleared by a dose of calomel or blue pill. Sodium salicylate or potassium iodide or both together may be given with advantage, especially in cases occurring after rheumatism or influenza. If an abscess forms it must be evacuated either by means of a trocar or by incision and drainage.

TUBERCULOSIS.

Tuberculosis of the thyroid gland is a rare disease, but it appears from facts recently collected (P. Bruns [188]) that it is not so rare as was generally supposed. In 100 post-mortem examinations on persons who had suffered from tuberculosis, Chiari found that the thyroid gland was involved in 7; 96 of the cases had suffered from chronic tubercular phthisis, and in only 4 of these was there tuberculosis of the thyroid gland; the other 4 were cases of acute tuberculosis, and in 3 of these the gland was affected. Tuberculosis of the thyroid gland may occur as miliary tuberculosis or there may be large caseous tuberculous nodules. The former does not give any distinctive signs, but the caseous nodules may be sufficiently large to form a goitre. Bruns [188] mentions six examples of tuberculous goitre.

HYDATID DISEASE.

Hydatid cysts may occur in the thyroid gland as in other parts of the body and lead to considerable enlargement of the gland. Semon [187] refers to two cases in which perforation of the trachea occurred and was followed by death.

SYPHILIS.

Engel-Reimers [189] has found that some swelling of the thyroid gland is very common in early syphilis and that it may persist for some time. He found such swelling in fifty per cent. of the cases he examined. Distinct syphilitic disease of the thyroid gland is very rare—in fact, I have found the record of only one case in which this disease of the gland appears to have been sufficiently extensive to cause myxœdema. In this case, recorded by Köhler,[190] the myxœdema disappeared under the influence of antisyphilitic treatment. Gummata are rarely seen in the thyroid gland (Ziegler [191]).

ACTINOMYCOSIS.

One case has been observed by Köhler [192] in which actinomycosis of the thyroid gland interfered so much with its function that the patient developed myxœdema. After suitable surgical treatment the actinomycosis was cured and the myxœdema disappeared. In such a case it might be as well to try the internal administration of potassium iodide, which has proved so serviceable in the treatment of actinomycosis, before resorting to surgical measures.

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